THE JOURNAL OF PERIODONTOLOGY

January
1949

Published by
THE AMERICAN ACADEMY OF PERIODONTOLOGY
VOLUME 20.
NUMBER 1.

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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, those papers of scientific and clinical interest which are presented at the annual meetings of that society.

Ninth Postgraduate Seminar

THE AMERICAN ACADEMY OF PERIODONTOLOGY

May 2, 3, 4, 1949

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Conservative Technic According to Dr. John M. Riggs

"THE third stage presents greater difficulties from the greater depth of the active line of disease, and demands a firm and skilled hand, a delicate and nice touch, and, I might add, the transfer of the sense of sight to the fingers ends. The sense of touch must be so trained and cultivated that all foreign bodies can be readily distinguished from tooth substance—live bone from necrosed bone—healthy from diseased tissue. This manipulation cannot be attained at once, but time and practice, with close and earnest study, will qualify and school the hand, and embolden the true and sensitive mind to achieve success in the treatment of this third stage."

—Reprinted from
Annals of Dentistry 7 (2):65, June 1948
Extract from "Eponyms: Riggs' Diseases"
By Lester R. Cahn, *New York, N. Y.*

THE JOURNAL OF PERIODONTOLOGY

JANUARY, NINETEEN HUNDRED FORTY-NINE

The Experimental Basis for the "Bone Factor" Concept in Periodontal Disease

By IRVING GLICKMAN, B.S., D.M.D.* *Boston, Massachusetts*

INTRODUCTION

IT IS generally recognized that information regarding the bone destruction responsible for tooth loss in periodontoclasia** is a basic need in the periodontal field. In the past, this problem has been approached from the following two aspects: the first deals with causation, particularly in regard to the relative significance of local and systemic factors; the second, with the actual microscopic changes which constitute the bone destructive process.

In the question of causation, the following major theories have evolved: 1. Bone destruction is a secondary feature of periodontoclasia which is the result of local factors alone (1); 2. Bone destruction is the primary change in periodontoclasia and is in all cases initiated by systemic factors (2), (2a); 3. Bone destruction may be either the primary or secondary phase of periodontoclasia and may be caused by either local or systemic factors alone or may be of combined local and systemic origin (3).

The microscopic changes responsible for alveolar bone loss have been described under various experimental conditions (4), (5), (6), and in different autopsy studies (7), (8), (9). Instead of establishing a clearly defined concept regarding the modus operandi of the bone destructive processes in periodontoclasia, the diversity of opinion which arose from such studies was indicative of the need for further clarification of this important problem.

THE "BONE FACTOR" CONCEPT

In an effort to explore the problem of bone loss in periodontal disease, a combined autopsy and experimental project was undertaken in 1938. From this project there evolved the "Bone Factor" concept. The present paper is offered in explanation of this concept on the basis of the evidence from which it was formulated.

The initial phase of the project consisted of a microscopic study of human autopsy material (10). Correlation of the observations in alveolar bone and other bones of the skeletal system led to several conclusions regarding the physiology of alveolar bone, in so far as could be evaluated by microscopic appearance alone. No morphological basis could be detected for the differentia-

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**The term "periodontoclasia" is used here to connote a disease process in which mobility, migration, elongation and tooth loss result from destruction of the supporting tissues. It is intended in a non-specific sense, and is used instead of such terms as "schmutz pyorrhea", "diffuse alveolar atrophy", "periodontosis", "periodontitis", or "syndrome," in order to avoid the specific inferences regarding causation frequently associated with the latter terms.

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tion of the bone adjacent to the periodontal membrane from bone in relation to periosteal surfaces elsewhere in the body. All of the changes exhibited by bone adjacent to the periodontal membrane and unrelated to the gingival margin could be duplicated along the surfaces of bone trabeculae in the remainder of the jaws and other bones.

PERIODONTOCLASIA NOT A SPECIFIC TYPE OF BONE DISEASE

Bone loss in periodontoclasia was shown to occur in different ways. None of the resorptive changes responsible for the bone loss were peculiar to alveolar bone. All could be demonstrated elsewhere in the skeletal system. In individual cases, bone loss might result from a combination of resorptive processes. It was inferred, therefore, that periodontoclasia was not a specific type of bone disease and that it could not be classified into different types on the basis of microscopic bone changes alone.

It was also observed in human tissue that alveolar bone loss occurred either associated with gingival inflammation or without it. When such bone loss occurred in the absence of gingival inflammation, it resulted from a predominance of bone resorptive over bone formative changes. In those cases in which there was a predominance of bone resorption in the periodontal area in the absence of gingival inflammation, bone resorption was also more prevalent than formation in the remainder of the jaws and in other bones.

RELATION OF GINGIVAL INFLAMMATION TO BONE LOSS

When present, gingival inflammation was interpreted as contributing to the underlying bone destructive process. Osteoclastic activity, a normal microscopic feature of alveolar bone was increased in zones of gingival inflammation. Although bone resorption was the more obvious finding in areas of inflammation the presence of bone formation in close proximity to areas of resorption was also noted. The presence of both bone resorption and bone formation in such areas indicated that the bone loss instead of being a simple destructive process actually represented the resultant of a predominance of bone resorption over formation.

Considerable significance was attributed to the fact that cases of periodontoclasia in which radiographic evidence of bone loss was a significant clinical diagnostic sign revealed a concomitant occurrence of bone formation and resorption associated with gingival inflammation upon microscopic examination. The presence of bone formation as well as resorption in areas of inflammation suggested that the destructive effect of the inflammation might not be the sole determinant of the severity of the underlying bone loss. By replacing, in part at least, the bone destroyed, the bone formed in such areas might diminish the total resultant bone loss associated with gingival inflammation.

Since the formation of bone in relation to areas of gingival inflammation could restore bone structure apparently destroyed by osteoclastic activity stimulated by the inflammation and since the ability to form alveolar bone was correlated with the degree of osteogenic activity prevalent in the remainder of the skeletal system, then the total bone loss which occurred in relation to areas of inflammation, might in some degree depend upon an intrinsic systemic bone tendency which governed the relative resorptive and formative activity throughout the body. It was therefore felt that the initial autopsy study pointed to the likely existence of a systemic regulatory influence which governed the severity of bone loss in periodontoclasia both in the presence and absence of gingival inflammation.

However, because autopsy material presents only the terminal static picture of disease, it is of limited value as an indication of causal relationships. It is, therefore, not possible to determine from such material alone whether the bone resorption associated with gingival inflammation is the result of the inflammation or the cause of it, or the extent to which systemic influences may be responsible for either or both of these findings. It seemed that the latter information could come only from animal experiments in which, by controlling both local and systemic condition, an effort could be made to determine the following: (a) whether altering the systemic background would effect the nature of the bony changes beneath areas of gingival inflammation; (b) what the nature of the effect upon the bone would be (c) how it would influ-

Fig. 1. Normal diet animal; head of tibia below epiphyseal cartilage showing bone containing remnants of cartilage and bordered by pale zone of new bone matrix. (Hematoxylin and eosin; orig. mag. x96.)

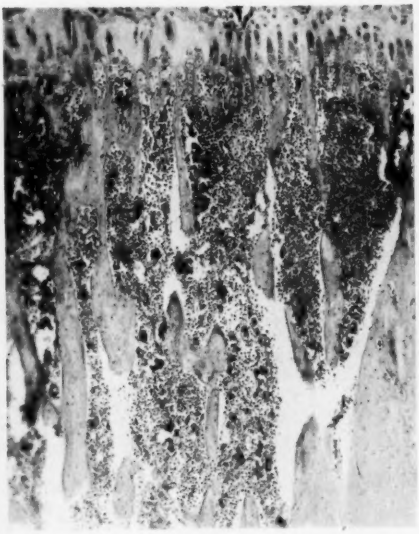

Fig. 2. Starvation animal; head of tibia below epiphyseal cartilage showing bone trabeculae devoid of bordering zone of pale new matrix and presenting roughened and fragmented margins. (Hematoxylin and eosin; orig. mag. x96.)

ence the severity of bone destruction arising from gingival inflammation.

ANIMAL EXPERIMENTS

Such a series of experiments was conducted. Amalgam was inserted between the mandibular first and second molars of albino rats in order to induce gingival inflammation. The animals were then divided into two groups, one of which was placed on a starvation diet and the other permitted unlimited food and water. Under these experimental conditions it was possible to study the effect of gingival inflammation alone upon alveolar bone; the effect of severe systemic disturbances such as starvation on alveolar bone, as well as the entire skeletal system; and the effect, if any, of a severe systemic disturbance such as starvation upon the nature of alveolar bone changes arising from gingival inflammation.

The following conclusions were derived from the experiments:

I. *In the absence of gingival inflammation, bone loss may occur in periodontal disease as a localized feature of a generalized skeletal disturbance of systemic origin.* The bone of the jaws undergoes changes similar to those which occur in other bones as a result of such a disturbance. Osteoporotic changes occurred in the tibia (Figs. 1, 2), rib, and vertebrae as well as the mandible of all animals on the starvation diet. Reduction in the height of the interdental septa subjacent to the gingival crevice was also seen in the starvation animals in the absence of gingival inflammation. This occurred because the physiological balance between bone formation and resorption, which determines the height of the interdental bone (Figs. 3, 4) was altered throughout the skeletal system so that bone resorption was predominant, and bone formation markedly reduced. The bone loss in the starved animals in the absence of gingival inflammation appeared microscopically as fragmentation and dissolution of the alveolar bone matrix with a release of osteocytes and the formation of connective tissue in areas previously occupied by bone (Figs. 5, 6, 7). Osteoclasis was not a prominent finding.

Because reduction in the height of alveolar

Fig. 3. Normal diet animal; interdental septum in the absence of inflammation, showing resorption lacunae and osteoclasts on the mesial side (right), pale new bone matrix with osteoblasts at distal (left) and border of new bone matrix at gingival crest. (Hematoxylin and eosin; orig. mag. x160.)

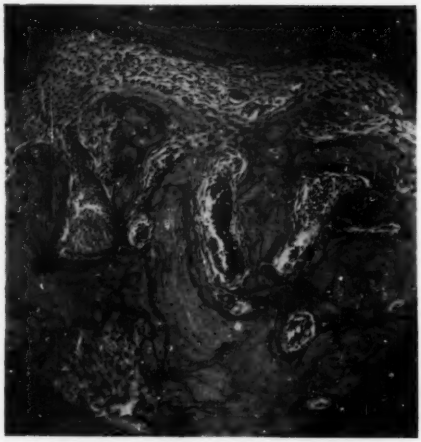

Fig. 4. Normal diet animal; bone in bifurcation of molar roots showing pale pink zone of bone lined with osteoblasts beneath cementum. The underlying trabeculae are for the most part lined with pale bone matrix. A portion of the trabecular borders is formed by continuous irregular concavities. (Hematoxylin and eosin; orig. mag. x146.)

bone is a cardinal clinical and radiographic sign of periodontoclasia, its occurrence as the result of a systemic disturbance in the absence of local inflammation was considered significant. It afforded some explanation for the many cases of periodontoclasia which present marked bone loss radiographically in the absence of clinically detectable gingival inflammation.

II. *Gingival inflammation alone may result in alveolar bone loss in the absence of a generalized skeletal disturbance.* The microscopic picture of bone loss beneath artificially induced gingival inflammation in the well-fed animals was comparable to that observed in human autopsy material. The bone margin beneath the area of inflammation was formed in part by irregular concavities of resorption and in part by a smooth, pale eosinophilic layer of bone bordered by a row of polyhedral osteoblasts (Figs. 8, 9). The underlying bone in the remainder of the septum was unaltered.

The fact that bone loss resulted from artificially induced gingival inflammation alone, in the absence of a generalized skeletal disturbance could not be interpreted as indi-

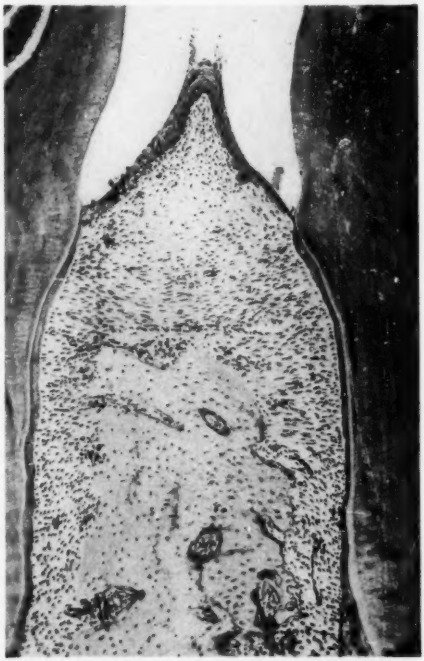

Fig. 5. Starvation animal; interdental septum in absence of inflammation showing resorption lacunae at mesial (right) and fuzzy remnant of pale pink matrix at distal (left) which blends with adjacent connective tissue. The gingival crest presents fragmentation of the bone matrix with loss of bone substance. (Hematoxylin and eosin; orig. mag. x80.)

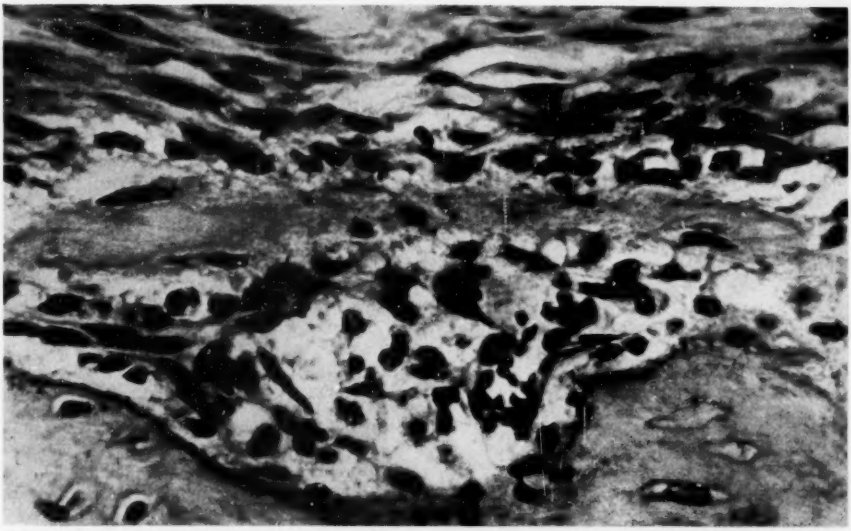

Fig. 6. Detail of Fig. 5; gingival crest showing fragmentation and dissolution of bone matrix and enlarged lacunae devoid of osteocytes. (Hematoxylin and eosin; orig. mag. x720.)

Fig. 7. Starvation animal; bone in bifurcation of molar roots showing peripheral resorption and fragmentation of bone matrix. There is loss of bone substance and thickening of the periodontal membrane with absence of bone formation (Compare with Fig. 4.) (Hematoxylin and eosin; orig. mag. x140.)

Fig. 8. Normal diet animal; interdental septum subjacent to area of artificially induced inflammation, showing infiltration of lymphocytes in relation to bone margin. (The localized accumulation of polymorphonuclear leukocytes immediately below the gingival epithelium is a reaction of the presence of a small bone fragment.) (Hematoxylin and eosin; orig. mag. x98.)

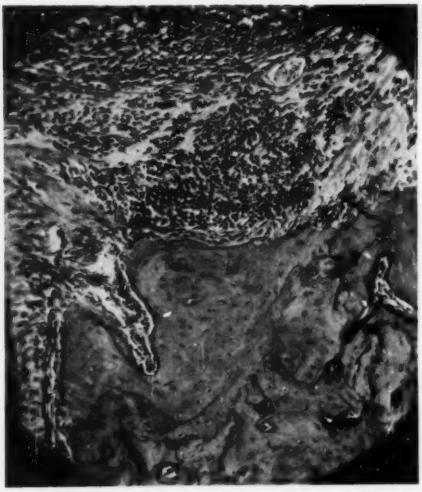

Fig. 9. Detail of Fig. 8; margin of interdental septum subjacent to area of artificially induced inflammation showing area of lacunar resorption (right) adjacent to new bone matrix lined with layer of osteoblasts (left). The underlying bone is unaltered. (Hematoxylin and eosin; orig. mag. x180.)

cating that it was a purely local phenomenon independent of the influence of prevailing skeletal tendencies. Actually, the influence of the prevailing skeletal tendency in the well-fed animals could be observed, microscopically, as newly-formed bone adjacent to the concavities of bone resorption. Such bone formation constituted a local manifestation of the over-all individual bone forming capacity.

III. *The severity of bone loss subjacent to gingival inflammation is increased by generalized skeletal disturbances of systemic origin.* The findings showed both quantitative and qualitative differences in the appearance of the bone beneath the inflammation in the starved and well-fed animals. The well-fed animals presented bone formation in close proximity to bone resorption in areas of bone loss beneath gingival inflammation. No comparable bone formation was observed in the starved animals (Figs. 10, 11). Because of the inability to form new bone throughout the entire skeletal system of the starved animals, the amount of alveolar bone loss in this group was greater.

EXPERIMENTAL DIABETES

Another opportunity to evaluate the effect of systemic disturbances upon the periodontal structures presented itself with the introduction of the drug Alloxan for the production of experimental diabetes mellitus (12). Examination of the periodontal structures in experimental diabetes revealed a tendency toward varying degrees of nonspecific osteoporosis of alveolar bone unrelated to gingival changes in approximately 40 per cent of the diabetic animals. These animals presented a reduction in the distribution and the amount of bone formation, an increase in bone resorption and a loss of bone substance associated with peripheral fragmentation and dissolution of alveolar bone matrix. (Figs. 12 to 17). All the bone changes occurred in the absence of gingival inflammation.

The loss of tooth supporting bone in experimental diabetes appeared as a local manifestation of a generalized tendency toward osteoporosis which affected not only the jaws, but also the remainder of the skeletal system. The findings re-emphasized the possibility that in periodontal disease neither the incidence nor severity of alveolar

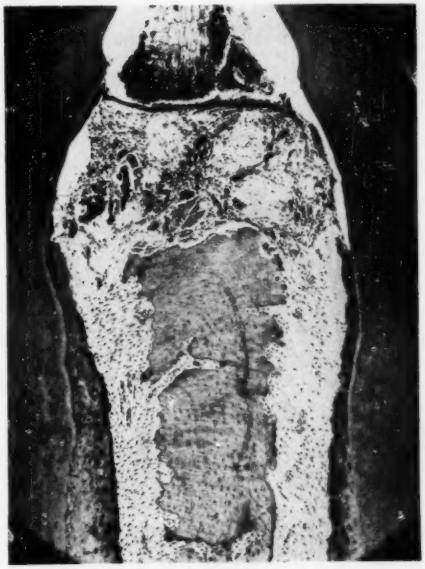

Fig. 10. Starvation animal; interdental septum subjacent to area of artificially induced inflammation showing chronic inflammation in connective tissue above bone margin. There is an absence of bone formation in relation to the entire septum. (Hematoxylin and eosin; orig. mag. x90.)

Fig. 11. Detail of Fig. 10; margin of interdental septum subjacent to area of artificially induced inflammation. The entire bone margin is formed by shallow concavities. For a considerable depth below the margin, the osteocyte lacunae are enlarged and empty. Fragments of bone are seen in connective tissue. There is no evidence of bone formation. (Hematoxylin and eosin; orig. mag. x150.)

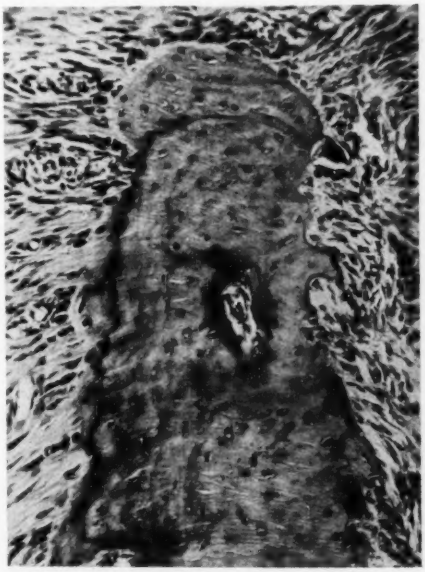

Fig. 12. Control animal; interdental septum of alveolar bone showing bone formation along mesial aspect (left) and along the crest of the septum. Resorption lacunae are seen along the distal aspect (right). (Hematoxylin and eosin; orig. mag. x240.)

Fig. 13. Control animal. Alveolar bone in bifurcation of molar roots. Bone deposition is seen adjacent to the periodontal membrane. The vessel channels and marrow spaces are lined in part by newly formed bone and in part by resorption lacunae. (Hematoxylin and eosin; orig. mag. x150.)

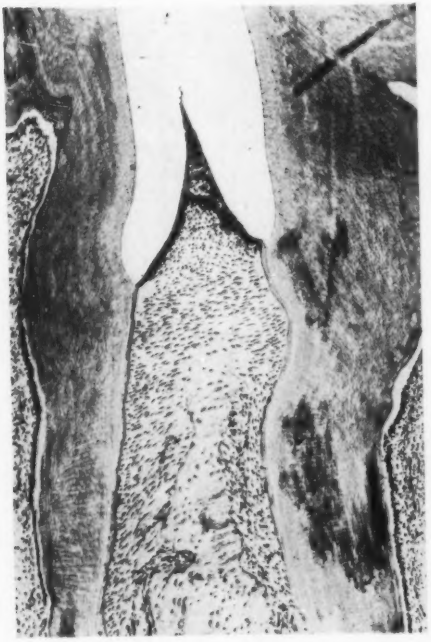

Fig. 14. Diabetic animal. Gingival papilla and underlying interdental septum of alveolar bone. The interdental septum shows an absence of bone formation along the mesial aspect (left) and crest where it is seen in the control animals. (Hematoxylin and eosin; orig. mag. x120.)

Fig. 15. Detail of Fig. 14. Crest of interdental bony septum showing absence of bone formation, peripheral fragmentation of the bone matrix and enlarged osteocyte lacunae. (Compare with Fig. 12) (Hematoxylin and eosin; orig. mag. x860.)

bone destruction need of necessity be related to gingival changes.

ACUTE VITAMIN C DEFICIENCY

The study of experimental diabetes was followed by investigation of the effect of acute Vitamin C deficiency upon the periodontal tissues of the guinea pig (13). It was observed that in acute Vitamin C deficiency, the periodontal membrane presented edema, hemorrhage, and pronounced collagen degeneration (Figs. 18, 19). The bone throughout the jaws was osteoporotic. The vessel channels and medullary spaces were enlarged and eroded (Figs. 20, 21). There was no new bone formation or osteoblastic activity in relation to the tooth supporting bone. The over-all picture was one of generalized destruction of the periodontal tissues in the absence of any notable gingival inflammation or pocket formation.

The findings in acute Vitamin C deficiency were comparable to those observed in experimental diabetes in that they showed loss of tooth-supporting bone unrelated to gingival inflammation or pocket formation. The findings in the Vitamin C experiments, however, differed from those in diabetes in the nature of the bony changes and in the fact that whereas both the periodontal membrane and alveolar bone presented severe destructive changes in Vitamin C deficiency, only bone changes had been observed in experimental diabetes. Comparison of the findings in two experiments is of interest because it demonstrates first, that more than one type of systemic change may produce generalized periodontoclasia; second, that although generalized loss of tooth supporting bone may be a symptom common to different systemic disturbances, the detailed nature of the bone changes need not be the same; third, when a generalized loss of tooth supporting bone occurs as the result of a systemic disturbance, the bone is not necessarily the only tissue involved nor need

it be the tissue in which the destructive changes are originally manifested.

Having established the nature of the periodontal changes arising from acute Vitamin C deficiency, the next problem was to determine its effect, if any, upon the response of alveolar bone to gingival inflammation. Gingival inflammation was artificially induced in Vitamin C deficient and control animals (14). As a result, the bone margin beneath the inflamed gingiva in the control animals presented both osteoclastic destruction and osteoblastic activity with new bone formation (Figs. 22, 23). Comparable areas in the Vitamin C deficient animals, however, presented no new bone formation. Instead there was degeneration of a collagenous prebony matrix and osteoporosis of previously formed bone. Osteoblasts were rare (Figs. 24, 25). A prominent accumulation of connective tissue cells which separated the bony margin from the gingival inflammation and apparently gave rise to the osteoblasts in the control animals was absent in the Vitamin C deficient animals, (Figs. 26, 27). This retardation in

Fig. 16. Diabetic animal. Bone in the bifurcation of molar roots showing absence of bone formation adjacent to the periodontal membrane and along vessel channels and marrow spaces. (Compare with Fig. 13.) (Hematoxylin and eosin; orig. mag. x140.)

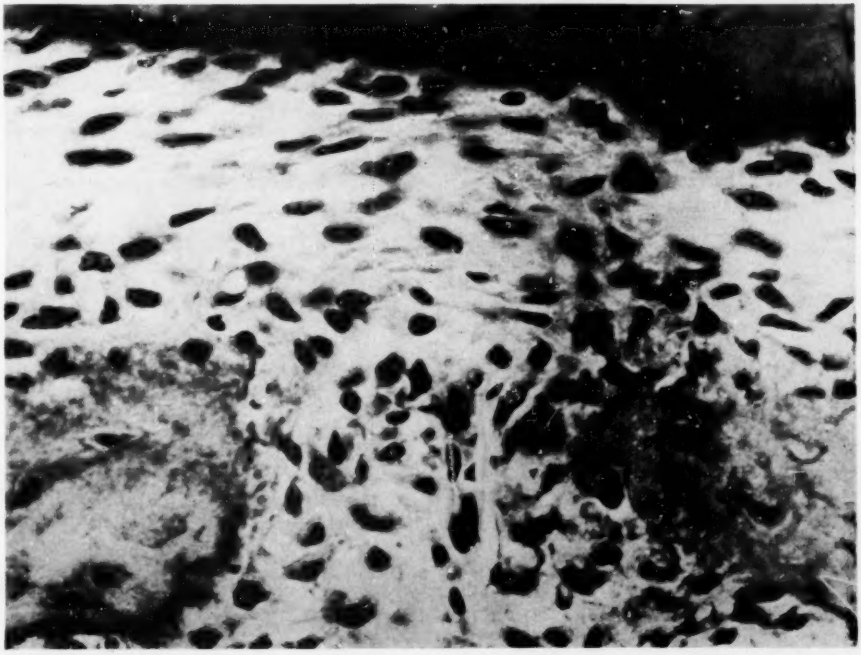

Fig. 17. Detail of Fig. 16, showing fragmentation of bone and release of osteocytes. (Hematoxylin and eosin; orig. mag. x780.)

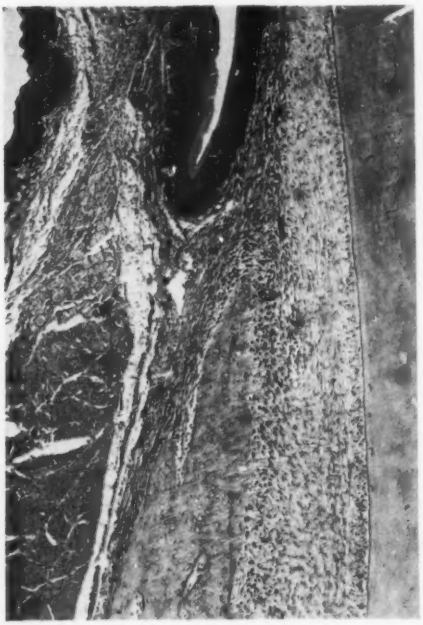

Fig. 18. Control animal, incisor area, lingual aspect showing division of fibres into a vertical group (adjacent to tooth) and a horizontal group (adjacent to bone). Note bone formation along lingual bony plate. (Hematoxylin and eosin; orig. mag. x96.)

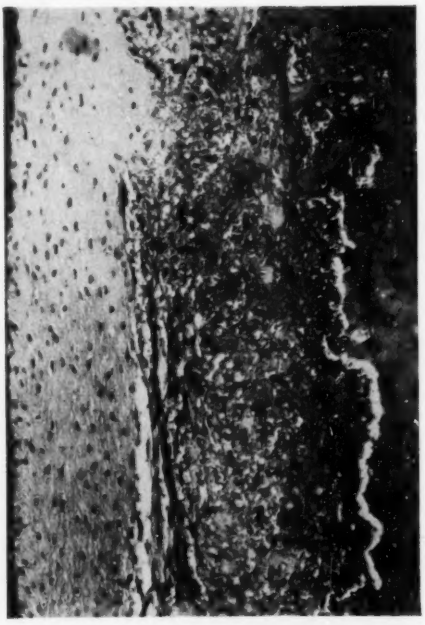

Fig. 19. Vitamin C deficient animal. Incisor area, lingual aspect, showing difference in the appearance of periodontal membrane. Adjacent to the bone (right) there is hemorrhage and marked degeneration of the collagen. Adjacent to the tooth (left) the collagen presents comparatively little degenerative change. Note eroded bone margin and break in the continuity of the collagen fibres and bone matrix on right. (Mallory's connective tissue stain; orig. mag. x150.)

the differentiation of connective tissue cells and ability to form and maintain new bone matrix accounted for the absence of well-formed new bone beneath the artificially induced gingival inflammation in the Vitamin C deficient animals.

The Vitamin C study added evidence in support of the existence of a systemic regulatory influence upon bone loss in periodontoclasia which had been suggested by the previous findings. In addition, the Vitamin C study demonstrated the specific manner in which the deficiency effected the response of alveolar bone to gingival inflammation. It was noted that although acute Vitamin C deficiency of itself resulted in loss of alveolar bone; artificially induced gingival inflammation was an added destructive influence. The bone loss arising from gingival inflammation was more severe in the Vitamin C deficient animals because of an inability on the part of these animals to maintain the existing alveolar bone or form new bone to replace that destroyed by the inflammation.

DISCUSSION

The sensitivity of alveolar bone to generalized skeletal tendencies suggested in autopsy study and subsequently corroborated by animal experimentation was deemed to be of clinical significance. By demonstrating the actual reality of systemic participation in periodontoclasia in terms of specific microscopic bony changes both in the absence and presence of gingival inflammation, these studies indicated the futility of attempting to evaluate periodontoclasia in terms of local influences alone. Since in health and disease, alveolar bone reflects the prevailing status of the skeletal system, alterations in alveolar bone in response to external stimuli are constantly subject to a systemic regulating influence. It is the systemic regulatory

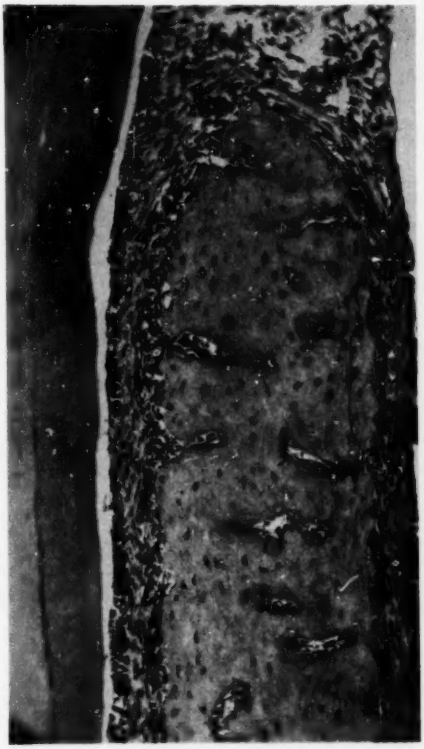

Fig. 20. Control animal. Interdental bony septum, molar, showing extensions from periodontal membrane into bone. Note osteoblasts along the mesial aspect of the bone (left) and osteoclasts along the distal (right). (Hematoxylin and eosin; orig. mag. x180.)

Fig. 21. Vitamin C deficient animal, interdental bony septum, molar area, showing edema and degeneration of periodontal membrane fibres. The bone margins are eroded throughout. The vessel channels are enlarged. There is no evidence of new bone formation. (Hematoxylin and eosin; orig. mag. x180.)

influence upon alveolar bone which has been termed the "Bone Factor" (15) in periodontal disease.

Clarification of the fundamental role of the "Bone Factor" in determining the nature of the bone changes in periodontoclasia is attempted in the following analysis:

Normally the height of alveolar bone is maintained by a constant microscopic equilibrium between bone formation and bone resorption. This physiological equilibrium is regulated by both local and systemic factors. In periodontal disease, bone loss occurs when the equilibrium is altered so that bone resorption exceeds bone formation. By stimulating resorptive changes in alveolar bone, gingival inflammation, pocket formation, trauma, and other local factors alter the microscopic balance between bone resorption and bone formation, and thereby initiate periodontal bone loss. However, the severity of bone loss which occurs as the result of local factors is not determined by their duration and severity alone. The response of alveolar bone to local destructive influences is subject to the additional regulation of the individual systemic background. Because it regulates the destructive effect of local factors upon alveolar bone, the "Bone Factor" is a basic determinant of the severity of bone loss in all cases of periodontal disease.

Two aspects of the "Bone Factor" con-

Fig. 22. Control animal. Area between base of periodontal pocket and underlying alveolar bone. Note concentration of inflammation beneath base of pocket. (Hematoxylin and eosin; orig. mag. x60.)

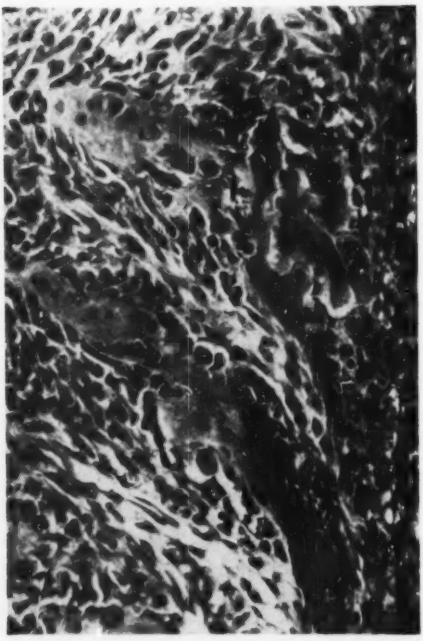

Fig. 23. Detail of Fig. 22. Tip of labial plate showing alveolar bone, new bone formation and osteoclasts. Note dense accumulation of connective tissue cells in area of new bone formation. (Hematoxylin and eosin; orig. mag. x380.)

cept are pertinent to the clinical problem of periodontal disease:

I. *The "Bone Factor" regulates the response of alveolar bone to the destructive effect of local factors.* Common to all of the local factors generally considered of causative significance in periodontoclasia is the fact that they produce inflammation in the gingiva and periodontal membrane. Gingival inflammation may be an early or late change. It may occur in otherwise unaltered periodontal tissues or it may appear in tissues already affected by destructive tendencies resulting from systemic influences. The significance of inflammation, regardless of when it occurs, lies in the fact that it stimulates destruction of alveolar bone. When inflammation is the initial periodontal alteration, its destructive effect in the presence of a normal "Bone Factor" is less severe than when it is superimposed upon an underlying bone destructive tendency of systemic origin. It may therefore be considered a general rule that the rapidity of the alveolar bone destruction which results from gingival inflammation will vary according to the status of the "Bone Factor."

Demonstration of the fact that the "Bone Factor" exerts a regulatory influence upon the destructive effect of gingival inflammation does not minimize the importance of local factors in the causation of periodontal disease. It merely indicates that the significance of comparable local factors varies from patient to patient according to the modifying effect of the individual "Bone Factor." It has been demonstrated above that the destruction of alveolar bone which results from a simple local gingival irritant may not be considered a purely local phenomenon independent of systemic regulation. Similarly, an alteration in the local functional environment which may be an innocuous or even bone formative stimulus in the presence of a favorable "Bone Factor," may become a local destructive agent when the "Bone Factor" is altered so as to inhibit the normal accommodation mechanism of alveolar bone (16). Because local factors are not the sole determinants of the severity of the bone destruction with which they are associated, evaluation of their significance in the diagnosis of periodontal disease or in the anticipation of the bone reparative activ-

ity which will follow their elimination requires judicious consideration.

II. *Periodontal bone loss may occur in the absence of harmful local factors if the "Bone Factor" is sufficiently altered.* The fact that periodontal bone loss may occur in the absence of gingival inflammation is not new to the periodontal field. It has been noted previously in clinical and autopsy studies and animal experiments. It is in large measure on the basis of such observations that a "systemic type" of periodontal disease has been described in which the loss of alveolar bone is attributed to systemic rather than local factors. Though there appears to be a unanimity of opinion regarding the likely existence of a "systemic type" of periodontal disease, there is some disagreement as to whether alveolar bone is actually the initial periodontal tissue affected in this condition. By the term "periodontosis" Orban and Weinmann (17), and Goldman (18), connote a condition in which the periodontal membrane is the tissue in which the earliest effect of systemic influences is manifested. Gottlieb (19) attributes the initial change in the "systemic type" of periodontal disease (diffuse alveolar atrophy) to the cementum. Box (20) describes "rarefying pericementitis fibrosa" as a "systemic type" of periodontal disease in which connective tissue changes constitute the early lesion.

Although the "Bone Factor" concept recognizes destruction of alveolar bone from systemic disturbances in the absence of harmful local factors, it does not support the arbitrary division of periodontoclasia into so-called "local" and "systemic" types. It conceives of the systemic influence upon alveolar bone as being constantly effective in health and disease. It is the nature of the systemic influence, in so far as it is reflected in specific microscopic changes in alveolar bone which varies. Because the status of the individual systemic background cannot always be catagorized by the extremes of "health" or "disease," its "formative" or "destructive" influence upon alveolar bone is of necessity characterized by subtle variations rather than by the opposite extremes suggested by attempts at a clear-cut clinical division of periodontoclasia into so-called "local" and "systemic" types.

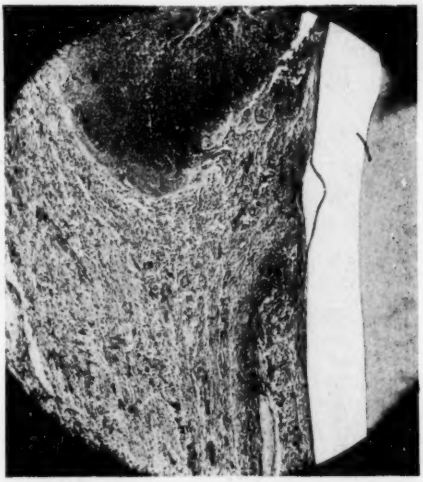

Fig. 24. Vitamin C deficient animal, labial surface of mandibular incisor, showing connective tissue and bone at the base of periodontal pocket. (Hematoxylin and eosin; orig. mag. x200.)

Fig. 25. Detail of Fig. 24. Tip of alveolar plate showing degenerated prebony collagen matrix adjoining osteoporotic alveolar bone. Note absence of osteoblasts and degeneration of surrounding periodontal membrane. (Compare with Fig 23.) (Hematoxylin and eosin; orig. mag. x380.)

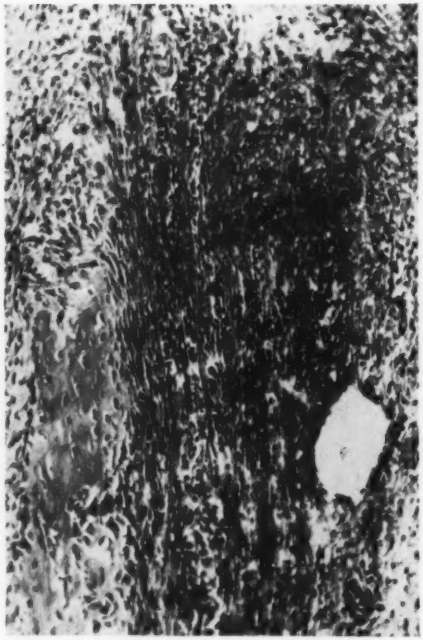

Fig. 26. Control animal. Tip of labial plate in close relation to inflammation. Note new bone formation and connective tissue cells which separate bone from adjacent inflammation. (Hematoxylin and eosin; orig. mag. x200.)

Fig. 27. Vitamin C deficient animal. Labial plate in close relation to inflammation. The entire tip of the labial plate is formed by degenerated collagenous prebony matrix. Note absence of peripheral accumulation of connective tissue cells. (Compare with Fig. 26.) Hematoxylin and eosin; orig. mag. x220.)

In healthy individuals, a positive "Bone Factor" or favorable bone formative potential exists. It is manifested microscopically by osteoblastic activity and well-formed new bone in the periodontal area as well as throughout the skeletal system. The bone formation described above in close proximity to resorptive changes in relation to gingival inflammation is evidence of the positive "Bone Factor" associated with systemic health. In the light of such microscopic findings it is difficult to presume that participation of systemic influences in periodontoclasia occurs only in unhealthy individuals or that such influence is of necessity detrimental to the maintenance of alveolar bone. Instead it would appear that there is a systemic component in all cases of periodontoclasia; that its nature varies in accordance with alterations in the systemic background, and that variations in the systemic component may retard or accelerate the bone loss incurred by harmful local stimuli or even result in periodontal bone loss in the absence of detectable destructive local factors.

In systemic disease an unfavorable or negative "Bone Factor" may exist. A negative "Bone Factor" means inability to form new bone or maintain that which already exists. Some systemic disturbances produce specific changes in alveolar bone (21), (22), (23). It has been demonstrated above that different systemic disturbances may be responsible for comparable non-specific destructive alveolar bone changes. All systemic diseases do not directly affect alveolar bone, nor is it only in specific systemic diseases that the "Bone Factor" may be altered. Skeletal osteoporosis has been described associated with modifications of physiological processes not categorized as specific disease entities (24).

The "Bone Factor" concept does not infer that alveolar bone is the tissue of origin in all cases of periodontoclasia, or that it is the only periodontal tissue which may be affect-

ed by systemic disturbances or that it is always affected by all systemic alterations. Involvement of periodontal tissues other than alveolar bone has been produced by systemic disturbances in experimental animals (25). In periodontal disease the periodontal tissue in which destructive changes first appear is determined by the nature of the responsible causative factors. It is as fallacious to assume that all cases of periodontal destruction originate in the same tissue as it is to postulate that there is only a single condition, local or systemic, capable of inducing periodontoclasia.

The "Bone Factor" concept envisions systemic participation in all cases of periodontoclasia. As a consequence the clinical approach to the diagnosis of individual cases of this disease does not entail the determination of whether "systemic" factors are responsible for the bone loss or whether it is of purely "local" origin. The fundamental clinical problem is that of determining the extent of systemic participation and whether its influence is a favorable or harmful one.

RECAPITULATION

A new concept is offered in explanation of the fundamental nature of bone loss in periodontoclasia. According to this concept, the bone changes in all cases of periodontoclasia whether slight or severe, localized or generalized, are constantly governed by what has been termed the "Bone Factor". The "Bone Factor" is defined as the systemic regulatory influence upon alveolar bone.

The concept is derived from autopsy study and animal experimentation which indicate that in systemic health and disease alveolar bone reflects the prevailing status of the entire skeletal system in terms of the relative predominance of bone formative or bone resorptive changes.

The "Bone Factor" constitutes the systemic component in all cases of periodontoclasia. Because it exerts a constant regulatory influence upon the response of alveolar bone to external stimuli, it modifies the destructive effect of gingival inflammation upon alveolar bone. When the individual bone formative capacity is good, a positive "Bone Factor" exists and the destructive effect of local factors upon alveolar bone is minimized. When throughout the skeletal system a tendency toward bone resorption exceeds the individual bone formative capacity, a negative "Bone Factor" exists and the destructive effect of local factors upon alveolar bone is exaggerated.

The existence of a negative "Bone Factor" may of itself be responsible for loss of alveolar bone, in the absence of local factors generally considered of causative significance in periodontal disease.

The "Bone Factor" concept discourages continued adherence to the clinical viewpoint which attempts to classify all cases of periodontoclasia as either purely "local" or purely "systemic" in origin. Such a viewpoint is inconsistent with the fact that the severity of bone loss which occurs in relation to even isolated zones of gingival inflammation is governed by a systemically motivated individual bone formative capacity. Since the status of the individual systemic background cannot always be categorized by the extremes of "health" or "disease", its formative or destructive influence upon alveolar bone is characterized by subtle variations rather than by the opposite extremes suggested by the clear-cut division of periodontoclasia into so-called "local" and "systemic" types.

REFERENCES

1. James, W. W. and Counsell, A.: "The Primary Lesion in So-Called Pyorrhea Alveolaris." *Brit. D. J.* 49:1129, 1928.
2. Hopewell-Smith, Arthur: "Pyorrhea Alveolaris. Its Patho-Histology." *D. Cosmos.* 53:397, April, 981. Sept. 1911.
2(a). Fleischmann, L. and Gottlieb, B.: "Contribution to the Histology and Pathogenesis of Pyorrhea Alveolaris." *D. Cosmos.* 63:215, 1921.
3. Orban, Balint: "Classification and Nomenclature of Periodontal Diseases." *J. Periodont.* 13:88, 1942.
4. Becks, H. and Morgan, A. F.: "The Effect of Deficiencies of the Filtrate Fraction of the Vitamin B Complex and of Nicotinic Acid on Teeth and Oral Structures." *J. Periodont,* 13:18, 1942.
5. Simonton, F. V. and Jones, M. R.: "Mineral Metabolism in Relation to Alveolar Atrophy in Dogs." *J.A.D.A.* 15:881, 1928.
6. Stones, H. H.: "An Histological Study of the Investing Tissues of the Teeth, the Dentin and Dental Pulp." *Brit. D. J.* 65:737, 1936.

7. Gottlieb, Bernhard: "The Formation of the Pocket: Diffuse Atrophy of the Alveolar Bone." *J.A.D.A.* 15:462, 1928.
8. Kronfeld, Rudolph: "The Condition of the Alveolar Bone Underlying Periodontal Pockets". *J. Periodont.* 6:22, 1935.
9. Simonton, F. V., "Etiology of Parodontoclasia." *J.A.D.A.* 14:1767, 1927.
10. Glickman, I. and Wood, H.: "Bone Histology in Periodontal Disease." *J. D. Res.* 21:35, 1942.
11. Glickman, I., Morse, A. and Robinson, L.: "The Systemic Influence Upon Bone in Periodontoclasia." *J.A. D.A.* 31:1435, 1944.
12. Glickman, Irving: "The Periodontal Structures in Experimental Diabetes." *N. Y. Journ. of Dent.* 16:226, 1946.
13. Glickman, Irving: "Acute Vitamin C Deficiency and Periodontal Disease. I. The Periodontal Tissues of the Guinea Pig in Acute Vitamin C Deficiency." *J. D. Res.* 27:9, 1948.
14. Glickman, Irving: "Acute Vitamin C Deficiency and Periodontal Disease. II. The Effect of Acute Vitamin C Deficiency Upon the Response of the Periodontal Tissues of the Guinea Pig to Artificial Induced Inflammation." *J. D. Res.* 27:201. April, 1948.
15. Glickman, Irving: "The 'Bone Factor' in Periodontoclasia." *Bul. Mass. D. Soc.* 20:14, 1944.
16. Glickman, Irving: "The Effect of Acute Starvation upon the Apposition of Alveolar Bone Associated with the Extraction of Functional Antagonists." *J. D. Res.* 24:155, 1945.
17. Orban, B. and Weinmann, J. P.: "Diffuse Atrophy of the Alveolar Bone. (Periodontosis)" *J. Periodont.* 13:31, 1942.
18. Goldman, Henry M.: "Periodontosis in Spider Montoclasia." *J. Periodont.* 17:7, 1948.
19. Gottlieb, Bernhard: "The New Concept of Periodonkey. Preliminary Report." *J. Periodont.* 18:34, 1947.
20. Box, Harold: "Studies in Periodontal Pathology." *Bul. No. 7. Can. D. Res. Foundation.* May, 1924.
21. *Op. Cit.* 13.
22. *Op. Cit.* 4.
23. Schour, I., Tweedy, W. R., and McJunkin, F. A.: "The Effect of Single and Multiple Doses of the Parathyroid Hormone on the Calcification of the Dentin of the Rat Incisors." *Am. J. Path.* 10:321, 1934.
24. Reifenstein, Edward, Jr., and Albright, Fuller: "The Metabolic Effects of Steroid Hormones in Osteoporosis." *J. Clin. Invest.* 26:24, 1947.
25. King, J. D.: "Abnormalities in the Gingival and Subgingival Tissues due to Diets Deficient in Vitamin A and Carotene." *Brit. D. J.* 68:349, 1940.

A Pattern of Centric Occlusion*

By J. C. WESTBROOK, JR., *Birmingham, Alabama*

CENTRIC JAW RELATIONSHIP has been defined as the most retruded position of the mandible from which lateral excursions can be made. There has been some discussion as to the possibility of retruding past true centric, but this is largely an academic question, for there is little danger of positioning the mandible too far distally.

It has been proved conclusively that centric closure is a true hinge movement. B. B. McCollum has designed an instrument which accurately duplicates all of the mandibular jaw movements and also enables the operator to definitely locate the hinge axis which is constant in each individual.

In a periodontal practice, it would seem that practically a hundred per cent of the patients examined have centric derangements to some extent. These vary from the slight premature contacts to the most extreme, such as caused by molar extrusion from loss of antagonists. This results in the loss of a rest position or free way space where the facial muscles are in equilibrium. There is also the eccentric intercuspation as described by McLean where the teeth—possibly a full complement of thirty-two—may be brought together in normal intercuspation only in an eccentric jaw relationship. This relationship may be a direct protrusion or may be slightly lateral as well as protrusive.

A complete examination should naturally include full-mouth roentgenograms in order to determine the condition and quality of the supporting structures, the length and shape of the roots. The weaker the support, the more nearly perfect should be the occlusion in order to minimize the strain on the weakened supporting structures. If there is

*Presented before the American Academy of Periodontology, at its Thirty-second Annual Meeting, Sept. 9, 1948, *Chicago, Ill.*

marked destruction of the periodontal tissues, a faulty occlusion is to be strongly suspected.

The clinical examination should consider primarily the type of occlusion from an orthodontic standpoint, the mobility of the teeth, and the balance in centric, lateral and protrusive relationships. In checking the centric relationship, first, have the patient relax the jaws, with the teeth almost touching. Then guide the mandible gently closed and check the relationship as compared to that which seems normal to the patient. From this position, have the patient retrude the mandible as far as possible with the teeth touching lightly. If there is a centric abnormality, it will readily be observed as there will be a marked opening of the bite as the mandible is retruded.

Another check is to have the patient close and attempt to make lateral excursions with firm pressure on the teeth. If this cannot be accomplished, it is usually an indication of inability to first close in centric.

Still another and very reliable check is to trim a piece of softened bite wax so that it covers only the posterior teeth, allowing observation of the anteriors when used. Have the patient bite into this gently with the mandible retruded, stopping before the teeth make contact. Repeat this until a thin area shows a very near occlusal contact. This can be used to check the premature contact and also mount study or record models. S. W. Brown has designed an "Analyzer" on which the casts are mounted and which demonstrates the centric abnormality very clearly.

The next phase of the examination is to check the type of bite and length of the cusps as shown in the lateral and protrusive relationships. It is possible to shorten cusps to a marked degree without adversely affecting the centric relationship or closing the bite more than is desirable. The bite is always being closed in correcting centric abnormalities, but only in centric relationship. Naturally, in a closed bite it would be unwise to shorten the posteriors more than might be absolutely necessary.

Before grinding, the patient should be informed that the teeth are being reshaped, not ground down flat, and that this procedure will not remove as much tooth structure as would be done by a diet such as nature intended.

A thin, heavily inked articulating paper is used to mark the contacting surfaces and frequently the paper is removed and the same closure is repeated. The areas where the carbon is removed by the second closure is accepted as the true registration.

The articulating paper is placed and the patient instructed as to how to close. One method of registering true centric contacts is to have the patient barely close "the back teeth" (without pressure enough to mark) then retrude and tap the teeth very gently and slowly. Sometimes it is helpful to use pressure to help hold the mandible back. If this is done and there is centric interference, which has been located previously, the marks will show up on the distal inclines of the lower and mesial inclines of the upper.

The best rule in grinding seems to be to preserve the cusps which will make centric contact in the fossae of the opposing teeth. This will eventually create reciprocal contacts both bucco-lingually and mesio-distally and create the best possible functional occlusion.

In order to simplify balancing in the excursive movements and in order to cause a more even wear, a system of ball and plane centric contacts are established. This means that each ball contact, which is always toward the end of the cusp, has its opposing plane contact toward the base of the fossa and which glides along these planes in the excursive movements of the mandible.

The ball contacts are shaped by grinding with a small stone from the fossa toward the end of the cusp to the point where the desired contact is located. The size of the ball contact is determined by the mobility of the tooth; the more mobility, the smaller the ball contact. This is done to cause an even amount of wear. Thus in a normal occlusion, considered from an orthodontic standpoint, the most important cusps are the mesio-lingual cusps of the upper molars, the disto-buccal cusps of the lower molars and the buccal cusps of the lower bicuspids.

As soon as there is sufficient correction in centric to allow the jaws to contact with a solid sound as contrasted to the original

muffled or chattering sound of the premature contacts, it is well to check in lateral and protrusive to determine the balance and uniformity of the planes. This is done before the bite is completely closed in centric in order to have as nearly an ideal balance as possible. In checking lateral, it is usually effective to have the mandible move in one lateral and back, then the other lateral and back as it is very difficult for the patient to move the teeth from side to side in true lateral excursions. As the bite is still being closed at this stage, the entire plane may be ground. It is not usually desirable to grind the ball contact at this point unless it rides clear out and over the end of the cusp of the opposing plane.

After the bite has been closed in centric to the point that it is at least level with the other positions (normal centric is the most closed position) by alternately working in centric and lateral, it is very important not to grind either the ball or plane centric contacts in balancing in the excursive movements. The same system applies to the protrusive as well as the lateral.

In occlusal correction one of the most important considerations is the elimination of food impaction between the posterior teeth. This is caused not only by open contacts, but also by uneven or worn marginal ridges, movement of the teeth under pressure, and the wedging action of the cusps. In relieving the wedging force, it is usually best to even up the marginal ridges, create the desired escape grooves and then, if necessary, shorten the opposing cusp.

In correcting a closed bite, it is not practical to achieve a complete balance in the extreme positions, either laterally or protrusively, but it is helpful to have the planes fairly straight to the end of the cusp or until the cusp is out of contact. It is also best to have a smooth transfer to pressure from one cusp to another. For example, if the ball contact of the lower cuspid rides over the end of the upper cuspid and then the contact is transferred to the incisors, it should be with a smooth glide, not riding over the end of the cuspid and coming down on the lateral with a bump. This can be accomplished by grinding the plane of the upper cuspid so it is a straight glide to the end of the cusp where the transfer is made.

In protrusive correction, as many planes of as many teeth as possible are brought into contact with the same ball and plane principle as the lateral correction.

There are times when both orthodontic and reconstructive work are needed to create the nearest approach to an ideal occlusion which would be the maximum number of teeth making contact simultaneously in centric relationship in such a manner as to enable the maximum number of teeth to remain in contact over the widest possible range of excursive movements.

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Nutrition and Dental Health

RECENT REVISIONS OF RECOMMENDED DAILY DIETARY ALLOWANCES

By DOROTHEA F. RADUSCH, D.D.S., B.A., M.S., *Minneapolis, Minnesota*

THE recent revision of the recommended daily dietary allowances (1) of the National Research Council contains a significant figure. That is the change in recommended calcium allowance for adults, which has been increased to one gram daily, from the previous 0.8 gram. Not long ago, the accepted standard was 0.68 gram.

What does this increase mean in terms of food? It means that the pint of milk is no longer a sufficient quantity to supply the daily quota. Instead, three cups are needed. Researches upon human adult subjects are the basis for this change. Careful balance studies have shown that calcium utilization is not as high as it had been postulated. The studies show that the retention or absorption of ingested calcium is an individual variable from 11 per cent to 32 per cent

(2), (3), (4). On corresponding intakes of calcium, women on self-chosen diets retained less calcium than those on well-selected basal diets. With calcium intakes of 1.2 grams, absorption of approximately 22 per cent was shown in the three studies cited. Significantly, an increased intake from 0.3 gram to 1.2 grams brought about an increase in retention of calcium but was not sufficient to permit storage by all subjects. The calcium status was reviewed in Section VII of the series on Nutrition and Dental Health in this Journal in July 1946 (5).

To meet the new calcium allowance, consumption of three cups of milk will supply 0.84 gram and the balance to complete the gram can be expected to be obtained from the other foods in the diet. There is no scientific evidence of any harmful effects from ingestion of this quantity of calcium daily. There need be no concern about development of hypertension or blood vessel calcification (6).

The recent researches on human subjects have furnished results which re-emphasize the interrelationships of dietary factors. It has been shown that vitamin C consistently improves iron utilization and furthermore that the effect of calcium on iron absorption is variable but favorable. When the phosphorus intake is low, the beneficial effect of calcium and of lactose on iron absorption is limited. It becomes very clear that no dietary requirement is an independent entity.

Other changes in the report of the National Research Council have to do with a reduction of *calories* and of *B-vitamins* for adults. For the sedentary man the reduction is from 2500 to 2400 and for the moderately active from 2500 to 2400 calories. It is stated that the calorie values are not applicable to all individuals, but rather represent group averages. For the individual, the allowance must be adjusted up or down to meet specific needs. The proper calorie allowance is that which over an extended period will maintain body weight at the level most conducive to well-being.

Present figures for thiamine, riboflavin, and niacin (nicotinic acid) are as follows:

	Calories	*Thiamine*	*Riboflavin*	*Niacin*
Man—154 lbs. (70 kg.)		*mg.*	*mg.*	*mg.*
Sedentary	2400	1.2	1.8	12
Physically active	3000	1.5	1.8	15
With heavy work	4500	1.8	1.8	18
Woman—123 lbs. (56 kg.)				
Sedentary	2000	1.0	1.5	10
Moderately active	2400	1.2	1.5	12
Very active	3000	1.5	1.5	15

A footnote states that, "the fact that figures are given for different calorie levels for thiamine and niacin does not imply that we can estimate the requirement of these factors within 500 calories, but they are added merely for simplicity of calculation. In the present revision, riboflavin allowances are based on body weight rather than calorie levels. Other members of the B-complex are also required, though no values can be given. Foods supplying

thiamine, riboflavin and niacin will tend to supply sufficient of the remaining B vitamins." For adults (except pregnant and lactating women) receiving diets supplying 2000 calories or less, such as reducing diets, the allowances of thiamine and niacin may be 1 mg. and 10 mg. respectively.

There is evidence that the male adult needs relatively little *iron*. The need will usually be provided for if the diet is satisfactory in other respects. The same is not true for women.

The need for supplemental *vitamin D* by vigorous adults leading a normal life seems to be minimum. For persons working at night, and for nuns and others whose habits shield them from sunlight, as well as for elderly persons, the ingestion of small amounts of vitamin D (400 I. U.) is desirable.

A suitable allowance of *water* for adults is 2.5 liters (2.7 qts.) daily. Most of this quantity is contained in prepared foods and beverages.

The *phosphorus* allowances should be approximately 1.5 times those for calcium. In general it is safe to assume that if calcium and protein needs are met through common foods, the phosphorus requirement also will be covered, because the common foods richest in calcium and protein are also the best sources of phosphorus.

The quantitative requirement for *folic acid* cannot be closely estimated from evidence now available.

There is little information available concerning the human requirement for *fat*. While a requirement for certain unsaturated fatty acids has been amply demonstrated with experimental animals, the human need is not known. Since foodstuffs such as meat, milk, cheese, and nuts, contribute fat to the diet, it is necessary to use separated or "visible" fats such as butter, oleomargarine, lard, or shortening only to supply 7-15 per cent of the total calories. The suggestion of 1-2 tablespoons of butter or substitute in dietary patterns is thus sufficient "visible" fat daily.

In general, the recommended daily dietary allowances are objectives toward which to aim in planning practical dietaries. The recommended allowances can be obtained with a good variety of common foods which will also provide other minerals and vitamins for which requirements are less well known.

The daily dietary pattern recommended is as follows:

1 serving meat, fish, or poultry—(legumes 1/week)
3 cups milk, 1 egg—at least 5 per week, 1-2 tablespoons butter or margarine
5 servings fruit and vegetables: 1 citrus fruit or tomato, 1 deep yellow or deep green, 1 potato, 2 other servings
4-6 servings cereal foodstuffs: bread, rice, macaroni, breakfast cereals, etc., at least one-half in whole grain form, the rest enriched
Desserts once a day unless calorie needs are high.

REFERENCES

1. Food and Nutrition Board, National Research Council. "Recommended daily dietary allowances". *Nutrition Reviews* 6:319, Oct. 1948.
2. Burrill, Lida: "The effect of calcium and lactose on iron absorption in normal adults." Doctorate Thesis, U. of Minn. 1947.
3. Leverton, R. and Marsh, A.: "One hundred studies of the calcium, phosphorus, iron, and nitrogen metabolism of young women." *Agri. Exp. Stat. Res. Bul.*, U. of Neb., 1942.
4. McCance, R. S., and Widdowson, E. M.: "Mineral metabolism of healthy adults on white and brown bread dietaries." *J. Physiology* 101:44-85, 1942.
5. Radusch, Dorothea, F.: Nutrition and Dental Health. VII. Mineral availability and utilization—summary of dietary needs and recommendations. *J. Periodont.* 17:125, July, 1946.
6. Kasson, C. M., and McCutcheon, A.: "Hypertension and calcium intake," *Lancet*, Nov. 30, 1946.

In Our Opinion

HOME TECHNICS FOR THE CARE OF THE TEETH

Initial Statement

By HAROLD J. LEONARD, D.D.S., B.A., *New York, N. Y.*

THE demonstrations of toothbrush technic at the recent Meeting of the American Academy of Periodontology illustrated the old proverb that, there are more ways to the wood than one. Methods vary from that of T. Sidney Smith as demonstrated by Dickson Bell, who uses a four row wide flat surface brush with soft natural bristles set close together, to that of Charters who demands hard bristles in a small brush head with bristles arranged in well-separated pointed tufts. By the Stillman method the brush is started well up on the gingiva and brought down over the tooth surfaces, jiggling, vibrating or shimmying as it is brought down. By the Charters method it is started on the tooth surface slightly pointed away from the gingiva and brought up against the gingiva using a small round and round stroke that causes the bristles to cover all labial or buccal surfaces and the embrasure areas and give a series of compressions to the gingiva. Most periodontists want the gingiva to get a series of compressions and releases without the slightest friction or abrasion. Harry Lyons and Virgil Loeb think massage is unnecessary to restore inflamed gingivae to health providing all causative factors (both local and systemic) are removed, cleanliness is maintained and the teeth are otherwise given good function. Many periodontists want to avoid any stroking of the gingiva with the bristle ends because abrasion tends to injury especially with a hard bristle brush, thence to gingival recession and grooving of the tooth surfaces. Merritt wants the gingival surface to be brushed in order to induce growth of the thickened dense corneous layer which is the normal response of skin to friction. Orban uses soft wood points to cleanse the proximal surfaces of teeth in which the interdental papillae have receded to leave these surfaces uncovered. Others decry the use of soft wood for such a purpose because of the danger of leaving microscopic wood splinters and fibers in the soft tissues and thus inducing further irritation.

It is time that these differences of opinion in the field of periodontology were resolved. They do harm because they are confusing to students in this field. When equally good results are obtained by two very different methods it is evident that the principles underlying are not those of the differences. As scientific students we must seek further for the basic principles underlying the several methods. When discovered these will undoubtedly lead to economies in time and effort formerly spent on details of technic which were believed essential but are now found to be extraneous.

It would seem that in the home care technic there are three objectives to be attained in the mouth. In this presentation we are not concerned with other phases of hygiene such as diet, nutrition, and general systemic freedom from toxins, allergies, and psychosomatic disturbances. Also it is assumed that gross

irritants such as calculus, bacterial slime, food impactions and traumatic stresses have been removed by the periodontal and general dental treatment in the office. What remains for the patient to do is to keep the exposed tooth surfaces and those immediately under the marginal gingiva clean, to reduce the chronic inflammation and promote health in the remaining gingival and periodontal tissues by stimulating vigorous circulation of blood, and to induce development of a thick heavily keratinized epithelium over the marginal gingiva and an intact epithelium in the sulcus. The end result should be a light pink, stippled marginal gingiva, flat in contour so as to come to a thin margin, which is without exudation, which is fairly insensitive to probing and which does not bleed with any type of manipulation short of cutting. Microscopic sections of such tissue should show a minimum of lymphocytes and plasma cells because such cells indicate inflammation, including continued faulty circulation and under oxidation.

The means to attain these ends should be as simple, convenient, and inexpensive of money and time as possible. And they must be as nearly fool proof as they can be. All of us have seen the extremely sore gingiva due to abrasion with a hard bristle brush, or the unsightly recessions and the deeply grooved tooth surfaces due to vigorous back and forth scrubbing with a toothbrush and dentifrice.

There is fair agreement that the toothbrush is an effective, cheap, and simple means of accomplishing the objectives in most mouths. In others in which proximal tooth surfaces have been uncovered or there are deep tissue losses between the teeth or between roots, or deep seated inflamed areas remain to be healed, additional means are necessary. Since the soft brush as used by T. Sidney Smith and Dickson Bell is effective in their hands and since the hard bristle brush is not fool proof but may and often does cause abrasion and recession it would seem that the advocates of the hard brush should concede that hardness is not essential. If the brush is to be medium or soft it will need more width so that the bristle mass may have the necessary density or body. It would seem desirable to have the bristles in tufts in order that the brush may be cleansed and aerated. The pointed tufts spaced about the distance apart of the teeth evidently are not essential. Whether natural boar bristles or plastic bristles are used also does not seem essential providing the plastic is so constituted that it maintains its properties in humid weather. Whether the brush head is flat, concave, convex, or has a longer tuft of bristles at the end is also probably not essential as good results seem to have been obtained with all of these forms although it is difficult to believe that a markedly convex brush can be applied very effectively to all tooth surfaces. A small brush head may be advisable in many mouths because the space within which the brush must work to reach the buccal and disto-buccal surfaces of the upper second and third molars may be limited.

For most mouths in which deep periodontal inflammations are not left remaining, the gentle pressure of the toothbrush used to cleanse the teeth thoroughly is probably sufficient to stimulate the blood circulation, which is the objective of massage. Certainly the bristles cannot be forced thoroughly into

embrasure areas sufficiently to clean off adherent microbic growth without compressing the gingiva to some extent. It is perhaps possible that special effort other than this to stimulate circulation by means of massage is only necessary in those practices where deep areas of impaired tissue are left without resection, with the expectation that restoration of health will gradually occur as irritants are kept off and the circulation is restored.

The idea that the gingiva needs a frictional stimulation to increase the keratinous coat is probably not important as so many dentists obtain ideally healthy gingivae without special effort devoted to this. It would seem wise not to stress or advise brushing of gingiva with bristle ends in view of the great harm that may come when this method is abused.

The great principle common to all methods of mouth care is cleanliness. The technic of home care must be sufficient to cleanse the teeth completely of bacteria, fungi, food, salivary deposits, and tobacco tar at sufficiently frequent intervals so that further irritation of the gingiva is prevented and adherent colonies of fungi and deposits of calculus are removed before they have become fixed. For most adults, there seems to be general agreement that twice a day brushing is advisable as less frequent brushing has proved inadequate. More than this is not generally considered necessary and whether before or after meals is unessential. Persons who are bothered with food retained around denture parts or between teeth where gingivae are receded may need some special after meals cleansing.

Evidently the exact method of holding the brush or rotating or vibrating with it is unimportant providing that the method used reaches all tooth surfaces not covered by gingiva, and does not do harm by piercing or abrading the gingiva and also providing that enough strokes are used everywhere so that all deposits are actually completely removed at each brushing. These deposits are not scrubbed off with less than ten or fifteen strokes in each area. The time required in brushing will therefore range between two and ten minutes depending on the ease with which all surfaces may be reached and cleansed. No one angle of holding the brush against the teeth will be sufficient. Deeply receded areas will require special tilting of the brush to reach them without abrading the gingiva on either side. Uncovered tooth surfaces on the distal of third molars also require special tilting of the brush as do tooth surfaces adjacent to gaps where teeth are missing.

The proximal surfaces of teeth in which the gingiva fills well the interproximal spaces are sufficiently cleansed by a brushing technic that forces the bristles into the embrasure areas. The more the interproximal gingiva has receded the less efficient the brushing becomes in reaching effectively all portions of the proximal tooth surfaces. Other methods of cleansing these surfaces are the dental floss or dental tape used by the patient as it is taught to the dental hygienist, the soft wood points, or smooth toothpick, and the rubber point attached to the tooth brush or given a separate handle. These agents used as cleansing devices are thin enough to reach and cleanse the tooth surfaces inaccessible to the toothbrush. Care must be used that the gingiva is not stabbed, cut or otherwise injured in the application.

The occlusal surfaces seldom need brushing as normally they are cleansed by the mastication of food three or more times a day. Teeth which do not occlude on the other hand may collect deposits and these should be removed by the brushing. Hirschfeld likes to start the brushing by working on the occlusal surfaces partly as a means of softening the bristles somewhat before they come in contact with the gingiva, and also because in their fresh stiff state they can be forced more effectively into the occlusal pits and fissures.

The lingual surfaces of the teeth must be brushed thoroughly without abrading the lingual gingiva. There does not seem to be as much difference of opinion regarding technic on the lingual as on the labio-buccal surfaces. Any method that cleans the tooth surfaces thoroughly here is acceptable providing it does not cause abrasion ulcers which are painful. Gingival recession and grooving of root surfaces from toothbrush abrasion are seldom appreciable on the lingual-palatal tooth surfaces.

The dentifrice does not appear to be very important. A fine polishing agent incorporated does help in the cleansing. If the grit is coarse enough to be appreciable between the teeth it will in time help to create abrasion concavities in the teeth. A mild alkalinity in the dentifrice seems desirable as hypersensitive spots become less sensitive if kept alkalinized night and morning. Otherwise the exploited components and virtues of tooth powders and tooth pastes appear to be selling points without scientific or clinical validity.

The idea of Rule that caries can be entirely prevented by the home care technic is not generally held. The caries due to acid fermentation on the tooth surfaces is greatly aggravated by bacterial growth stimulated by gingival exudate. The thorough toothbrush technic should remove all accessible bacterial colonies and stop gingival exudate. This will almost entirely prevent the inception and early progress of gingival third and root surface cavities. The cavities forming in faulty occlusal pits and fissures and under the contact points of teeth of children and young persons are not so accessible to a cleansing technic and are not readily controlled by this means.

I have never insisted on routine scrubbing or scraping of the tongue. If it is heavily furred or coated due to indigestion or other illness, or heavy smoking there may be an advantage to cleansing it in a better tasting and certainly a better smelling and cleaner mouth. Otherwise I see no advantage in it and there are some drawbacks such as the gagging induced in many individuals.

Some patients have injurious mouth habits such as pushing against the teeth with the tongue, biting objects or clenching the teeth (bruxism) for long periods either day or night. Such habits must be broken or injury will be caused to periodontal structures. Lack of function due to bolting of food or avoidance of food requiring vigorous chewing is probably equally injurious. Sucking lemons or oranges will cause erosions on the labial surfaces of the teeth. Such habits while important in the home care of the mouth need not be considered in the present discussion.

Our object is to bring out the essentials and unessentials in the home care technics of the mouth. If each of the advocates of special technics will examine

his technic in the light of the success others are having with other methods perhaps we may approach the essentials of the problem. It is so important that the leaders in the field of periodontology eliminate their glaring divergencies that each must sacrifice something of a vested interest in his own published technic to this end.

Discussion

STANLEY C. BAKER, D.D.S.
Greenwood, S. C.

In Chicago, at the meeting of the American Academy of Periodontology in September, I had the pleasure and privilege of seeing ably demonstrated three technics of tooth-brushing — namely, the T. Sidney Smith method, the Charters method and the Stillman method. All three methods have their merits, and since it is the prime effort of all of us to advise and to help patients keep their mouths clean, once we have removed all causes for uncleanliness, it is essential that we give instructions which will be easy to follow and yet will attain our ultimate goal.

The basic principle of each of the above mentioned methods is to properly cleanse the oral cavity by cleaning all exposed tooth surfaces, especially at the gingiva, thus promoting gingival tissue health. This of course includes the interproximal spaces as well as the tongue and cheeks.

There is a question in many minds as to the adequacy of the toothbrush, for the purpose of this cleaning. It is, however, the only implement so far designed that we can safely put into the hands of laymen to perform this most important duty. If we could then agree on a definite technic to be universally used, both patient and dentist would be spared much grief. Whether to recommend hard bristles, soft bristles, two-row, three-row, or tufted heads is the problem. In my opinion, the two-row hard brush serves best for interproximal stimulation and the three-row soft brush for the gingivae. Let me say here that we are sadly in need of a brush designed especially for the lingual surfaces.

It should not be our object to burden the patient with tedious technics and numerous types of brushes, but to encourage cleanliness by a technic so simple that we can start with children, and continue developing the technic to adult age, thus saving many worries in later life due to wrong methods of brushing over a period of years. My reason for advocating the *early* teaching of the correct use of the toothbrush is that, should periodontal lesions develop later in life from any other cause than uncleanliness, such as malocclusion, poor dentistry, or failure to see a dentist, the home technic will then not be so hard to master.

I realize that it will be practically impossible to adopt a single technic which will be acceptable to dentist and patient, but with the American Dental Association endorsing methods, toothbrush manufacturers offering some, and each dentist advocating what he feels is best, one can easily see how confusing it all is to patients. I *do* believe, however, that it is time to try to standardize toothbrushes and home care technics to such an extent that dentists can talk the same language to one another and to patients, thus combining all efforts to attain clean, healthy oral cavities through intelligent home care.

DICKSON G. BELL, D.D.S.
San Francisco, Calif.

Two statements in the first paragraph probably need further explanation and may not necessarily be two different schools of thought. Lyons and Loeb refer to massage of *inflamed* gingival tissue while Merritt wishes to develop a toughened corneous layer in *normal* gingiva.

In the third paragraph I would question the statement of the second objective of home care—"to reduce the chronic inflammation and promote health in the remaining gingival and periodontal tissues by stimulating vigorous circulation of blood". This again brings up the matter of brushing inflamed gingival tissue. Menkin states that inflammation is a manifestation of a cellular injury brought about by the presence of an irritant. If we accept this premise, then the reduction of inflammation, wheth-

er chronic or acute, would be one of the objectives of periodontal therapy and not that of home care.

The design of the toothbrush is a very important factor, for example, a two-row stiff bristle toothb-ush with pointed tufts requires an entirely different brushing technic than one would employ with a level trim four-row brush with soft or medium bristle texture. Those which are concave, convex or have several rows of longer bristles at either end of the brush head do not accomplish satisfactory results. The cleansing and aerating of the bristles is a matter of personal cleanliness and attention. It has not been a problem with that type of brush having many bristle knots set close together and four rows in width.

In a discussion with Dr. Charters on interproximal care, he brought up an important point about the long soft wood point. viz., that—"they cannot be used from the lingual." The instrument he advocates and the soft rubber cone are so shaped that they can be more easily inserted into the lingual interproximal areas as well as from the bucal and labial.

Reference was made to a statement by Rule that caries can be entirely prevented by the home care technic. There are certain exceptions, of course, but my observation has shown that there is a definite decrease of dental caries in adults who have periodic prophylaxis and who conscientiously follow a regime of oral hygiene. Even patients who previously had frequent dental caries have noticed it and called attention to the fact that long periods of time have elapsed with either no carious lesions or only an occasional one.

In general I would agree with Dr. Leonard's discussion of the subject of home care. The procedure that I teach my patients was discussed in the last issue of this Journal and there is no need of repetition.

Dr. Leonard is to be commended for his selection of this topic as it is a most important and timely one. The first and logical approach to this problem of differences of opinion on toothbrushing technics is a correlation of the different ideas toward some agreement on the design of the toothbrush, the texture and length of the bristles.

HARRY LYONS, D.D.S.
Richmond, Va.

Dr. Leonard's "Initial Statement" dealing with technics used in the home care of the teeth and their supporting structures brings an important problem into sharp focus. I should like to commend particularly the second paragraph of his statement.

I agree wholeheartedly with Dr. Leonard that too many of us are guilty of quibbling over trivia. In so many phases of periodontology there is general agreement on certain basic principles. These are often submerged and befogged by petty arguments over technics. I agree that the time has come to emphasize our points of agreement and place our disagreements on minor issues in their proper perspective. The prevailing argument over the variations in technics of toothbrushing is a case in point.

Dr. Leonard clarifies a befogged issue by his statement to the effect that the basic objective common to all methods of personal mouth care is the maintenance of cleanliness. This may be interpreted to include freedom from all types of irritation. It would be strange, indeed, if cleanliness and freedom from irritation could be effectively attained only in one way. The time, energy and thought given to debate on these methods could well be applied to the search for scientific truths yet to be discovered.

In our arguments over unessential details, I fear that we often degrade our professional stature by widespread promotional publicity inadvertently given to certain commercial ventures. There is, for example, the instance of a toothpick alleged by its vendors to have the miraculous ability to make "gums glow with health." Despite such unwarranted, unsupported, and ridiculous claims, dentists in great numbers have added their professional endorsement to such claims by advocating the use of this particular toothpick. The "dignity" of this brand of toothpick has been raised to such a high level that its trademark has now become a term of common usage in the scientific (?) vocabulary of many dentists. The use of this toothpick has been made a major issue by its advocates. This may be viewed as a source of professional embarrassment when viewed in the light of scientific evaluation. There are too many similar examples that may be cited.

Dr. Leonard's statement is a clarion call. Our profession and the public would profit greatly if we would search for and debate principles, and relegate lesser issues to their proper places in the scheme of our professional endeavors.

ARTHUR H. MERRITT, D.D.S.

New York, N. Y.

After reading Dr. Leonard's "Initial Statement," it must be evident to anyone that it is not so much the technic that counts in the brushing of teeth, as it is the efficiency and perseverance with which it is put into effect. In other words, it's the man behind the brush that makes the difference between success and failure.

While there may be one *best* way in which to brush teeth, it is doubtful whether there will ever be a unanimity of opinion as to which is the *correct* way. In general it may be said that any technic that efficiently cleans the teeth and provides the necessary stimulation to the gums and does this without injury to either the teeth or gums, will meet all requirements. I will not add to the existing confusion by outlining another technic for brushing teeth. We have too many already. Moreover, I would probably make no converts if I did.

That which is of first importance is, that the method employed shall be simple—one that can be easily understood and easily put into practice by the patient. To complicate it by too many technical details, is to invite failure.

With this in mind, the patient should be *carefully* instructed in the use of the brush by demonstrations in his own mouth. He should then be handed the brush and asked to repeat the operation. His shortcomings, if any, can then be pointed out to him. These instructions should be continued until he is reasonably proficient. The essential thing is that he *master* the technic and *persevere* in its practice. Here is where most failures occur. Therefore, having taught the patient the ways and means for caring for his mouth, it should be made clear to him that the responsibility is now his and not his dentist's.

Nevertheless, in spite of the most careful instructions in the art of mouth hygiene, there will always be some who never will be expert in the use of the toothbrush. Once when I commented on the lack of skill shown by a patient in brushing his teeth, he replied "I have never done but two things with my hands in my life—fed myself and put my clothes on." There is more than one such patient in every office.

DONALD MOSHER, D.D.S.

Kansas City, Mo.

The teaching of a method for the home care of the mouth is a difficult problem.

The following is an outline of some of the technics we use in our office for the care of the teeth.

To begin with, we teach patients to start brushing the teeth on the left side, both upper and lower. However, if the patient is left-handed we suggest starting on the right side. First we place the brush on the labial surface of the last bracket of teeth on the upper left side, with the bristles sloping away from the gums. Then we place the side of the bristles against the gums and press them in between the teeth. Now by using a small circular movement of the brush we massage the gums and clean the teeth. Afterward the brush is moved forward and around until the last bracket of teeth is reached on the right side. This same technic is carried out on the labial surfaces of the lower teeth.

Next, the brush is placed on the lingual surface of the last upper tooth on the right side, and is worked up against the gums for stimulation, and down on the tooth for cleaning. It is then moved forward a tooth at a time for stimulation and cleaning until the last tooth on the upper left side is reached. This technic is carried out on the lower lingual surfaces, except that the brush is pushed down first and then up.

A week or two after finishing our treatment we make it a point to have the patient come in for a check-up. This is very important so that we can see if he is carrying out our instructions. The check-up consists principally of having the patient demonstrate his home care technics. We advise round tooth picks where there are small spaces between the teeth, and rubber points for wider spaces. We advise a two-row, semi-hard bristled toothbrush.

B. ORBAN, M.D., D.D.S.
Colorado Springs, Colo.

The differences in brushing technics as demonstrated at the recent Academy Meeting by Drs. Radusch, Bell, and Charters were indeed striking. However, I don't believe that these differences are harmful to our discipline. No technic in any field of the healing arts is the same if used by different men. Dickson Bell certainly would not insist on his technic if he couldn't get the desired results, and of course, the same is true for Charters and for all of us. We don't use the same instruments in performing a prophylaxis or gingivectomy either, and the technic of post-operative treatment varies widely.

I completely agree with the objectives set up by Dr. Leonard as to what we should expect from a healthy gingiva. It should be pale pink with thin, smooth margins, pointed papillae, stippled attached gingiva, shallow sulci and no exudate.

In teaching home care technic, we must make individual differences according to the condition of the gingiva at the time, the age of the patient and his capability of performing what we ask him to do along with his willingness to coöperate. The best home care technic isn't worth anything if it is not carried out according to instructions. Some individuals need much more home care than others and many just do not realize how much they need to do to keep their mouths in a satisfactory hygienic condition. In many cases a combination of different technics and different instruments (toothbrush, soft wood points, toothpicks, pipe cleaners, dental floss, rubber-tipped stimulators) are needed to keep the mouth clean and the gingiva well stimulated.

Any method or instrument used incorrectly might lead to damage, but the hardest toothbrush used properly will not injure the gingiva or teeth. A toothpick used where the interdental papillae fill out the interdental space is not only superfluous, but injurious. But if used when the gingiva has receded leaving a space in which food remnants and bacterial plaques are accumulating, it is beneficial.

I am satisfied that different methods can lead to similar end-results. It depends on who is teaching it and who is performing it. An individual variation according to the case in question will be most satisfactory.

DOROTHEA F. RADUSCH, D.D.S.
Minneapolis, Minnesota

The first impression upon reading the "Initial Statement" is that home care methods now advocated are inconsistent and therefore dentists and patients are confused. However, upon closer inspection, it is noted that the moderator says that all technics have the same basic principle, "to cleanse the teeth completely of bacteria, fungi, food, salivary deposits, and tobacco tar at sufficiently frequent intervals so that further irritation of the gingiva is prevented and adherent colonies of fungi and deposits of calculus are removed before they become fixed."

If this statement be accepted, then the following is also acceptable: "Evidently the exact method of holding the brush or rotating or vibrating with it is unimportant providing the method used reaches all tooth surfaces not covered by gingiva, and does not do harm by piercing or abrading the gingiva and also providing that enough strokes are used everywhere so that all deposits are actually completely removed at each brushing."

In such event there is no real confusion in the advocated technics. The technic and brush type in each cited example are properly associated. The soft brush can be satisfactorily efficient used in a manner demonstrated by Bell, the hard plastic bristle is well adapted to Charter's intermittent method, but is too stiff for continuous motion of the brush in each placement. The chosen technic is an expression of the dentist's preference, and, is not a sign of fundamental conflict, since all obtain the same end-result, which is clean tooth surfaces. My preference for the hard or firm brush technic comes through my observations on patients who have used both hard and soft bristle brushes. I am convinced that most often those who use the hard brush, frequently with a special aid, have cleaner, healthier mouths. Hardness is certainly not "essential", but I find it more effective and practical for the time the average patient in a general practice will spend on brushing. Firmness of bristle is an advantage in brushes used for other cleansing purposes, and seems logically to be an advantage in a

toothbrush. Possibly the firm brush gives more soft tissue massage simultaneous with the cleansing.

True, there may be differences of opinion regarding the need for massage, but I doubt that anyone can cleanse the teeth satisfactorily without an incidental and accompanying gingival massage.

There are three reasons for use of aids called "stimulators", but which would be more properly named if they were called "cleansing instruments". In the first place, many patients are not too skillful in manipulation of a toothbrush regardless of method. For them it is easier, simpler, and more effective to cleanse proximal areas by use of a specially designed instrument.

Patients find posterior and lingual interproximals most difficult of access, thus an instrument so designed as to facilitate use in these areas is most desirable.

The second reason for use of a special aid, is because as stated initially, "the more the interproximal gingiva has receded the less efficient the brushing becomes in reaching effectively all portions of the proximal tooth surfaces". Thirdly, because interdental recession results in protected areas which receive no functional stimulation, other friction would seem desirable. It is possible too that special efforts for compressing the gingiva are useful in assisting the healing process. Exact information as to the significance of massage as such must come from carefully designed experimental studies.

R. W. RULE, D.D.S.
Glendale, Calif.

As usual, Dr. Leonard's "Initial Statement" is quite full and comprehensive and covers most of the points for discussion.

The variations in the conditions of different mouths are so great that the technic of home care must be hand tailored for each case with the exception of the comparatively few cases in which the arches are well formed, the teeth well contoured with tight, well-shaped contacts and with normal, firm triangular septal gingivae filling the interproximal spaces. In these cases, Dr. T. Sydney Smith's "physiologic" method of brushing the teeth is both ample and ideal. (*See J.A.D.A.* Vol. 27, June, 1940 p. 874.)

Dr. Smith also discusses some of the deviations from these ideal conditions, but the deviations are so numerous and various that each case becomes practically a law unto itself and the dentist or periodontist should study the individual case in that light and give the needed instruction to the patient.

In my judgment, many patients do more harm than good in using the dental floss. It is more difficult to use (with the proper care and technic) on one's self, even for a dentist, than to use on a patient. If intelligently and carefully used, it is, in some cases, a valuable aid, but the patient should be fully instructed and warned of the dangers in a careless use of the floss. If there is much open space from loss of septal tissue, there will be less danger of damaging the tissues and it is one useful adjunct in the cleansing of such proximal surfaces.

The use of stiff, hard bristles, I condemn in toto. They lack the necessary flexibility for the best brushing technics and are almost certain to damage the gingivae. Also if used vigorously with certain dentifrices they will abrade the surfaces of the teeth. Stiff nylon bristles are worse than natural bristles in these respects although I rather prefer the nylon bristles if of small enough diameter and sufficient length to give the desired softness and flexibility. The bristles should be of even length, presenting a flat surface, and with enough rows to give suitable body to the brush. The conditions in some mouths demand a small brush and again, some patients can use a larger brush and are not satisfied with a small one.

When the lower incisors incline sharply inward, a special brush will likely be needed for this area of the mouth. This may be made by taking an ordinary brush and cutting away the bristles even with the handle except the three or four cross rows at the end of the brush.

Where there is much recession or loss of the septal tissues, toothpicks are often quite desirable. I prefer a well-made round toothpick with tapering round point at each end, used with an up-and-down movement on the proximal surface, carrying a dentifrice if desired, or, the flat wooden picks. The patient should be taught how and when to use these. I have not found soft wood points or the rubber interdental stimulator very

satisfactory. In a few cases of deep interproximal spaces difficult of access, the rubber stimulator may have a place.

Each patient should be advised to mentally visualize the form of the arches and the relationship of the teeth in the various parts of the mouth and keep this before them as they work in each locality, endeavoring to cleanse as thoroughly as possible every surface that can be reached. I have seen marked improvement when this instruction has been followed.

It is my judgment that many patients place too much confidence in the dentifrice and do not attach enough importance to thoroughness in the brushing. The use of a very mild dentifrice once every other day with thorough brushing twice daily should be ample for non-smokers. Smokers will need a somewhat more abrasive dentifrice and may need to use it every day. My preference for brushing times is after breakfast and sometime between the last meal of the day and bed-time. Many persons will feel the benefit of a thorough rinsing of the mouth before breakfast.

I believe that massage is unnecessary to restore inflamed gingivae to health providing all causative factors are removed for when this is done, inflammation rapidly subsides.

In reading the "Initial Statement," I observed with some surprise, the remark, "The idea of Rule that caries can be entirely prevented by the home care technic is not generally held." So far as I am aware, the statement as it stands, unqualified, is not held by Rule himself. The nearest thing I am able to find to such a statement in a hurried review of my writings is the following quotation with the underscoring added at this writing.

"The very best patients in one's practice are those who have been taught to realize the value of preventive measures, *both those which they can apply themselves* and those which may be *employed in service to them.* In addition, those whom they refer for a like service become the same type of patient. Many cases could be cited of patients who have been carried over periods of from fifteen to twenty years and longer with practically no disease of the mouth and teeth simply because of employment of *such measures* and *such service;* and there seems no reason that this condition should not continue through the remainder of their lives." (*J.A.D.A.* Vol. 20, July 1933, p. 1256.)

The above refers, of course, to the combination of office service and home care. Perhaps very few patients would accomplish these results without periodical prophylactic attention at the office.

I quote also from an article on "Teaching Methods in Oral Hygiene." "We teach that mouth hygiene, when well carried out by *both patient and operator,* will definitely prevent gingival diseases even in the face of a moderate handicap in the way of faulty nutrition and metabolism, but in *many cases* the *combined efforts* of patient and operator will *fail* in *complete inhibition* of *caries* . . . unless the patient will give unusually intelligent and thorough care." (*Proceedings of Amer. Assoc. of Dental Schools,* 1931.)

My present views after fifteen years more of experience and observation are not greatly different from these. It is, however, astonishing how far a coöperative patient can go in preventing disease, both of the teeth and their supporting structures, once the mouth has been put in first class condition.

T. SYDNEY SMITH, D.D.S.
San Francisco, Calif.

In the "Initial Statement," Doctor Leonard has given a good description of the various types of toothbrushes that are advocated for the daily care of the teeth and their investing tissues and the exact way they are to be used. These brushes have bristles that range all the way from the coarse, stiff nylon type that are to be forced into the interdental spaces with considerable pressure, to the fine, flexible natural bristles that are guided over the gingival tissues with gentle strokes that follow the same direction that the food does during mastication. Therefore, Doctor Leonard states that it is time that these differences of opinion in the field of periodontology were dissolved. I am sure that all will agree with him.

As I am the one who has advocated the physiologic method, the gentle technic, I have been requested to take part in the discussion.

I believe the most accurate way to reach a correct decision is to study the anatomic

form of the investing tissues of the teeth that permits the masticated food to glide harmlessly over them and turn the friction of it into a cleansing and stimulating action. To provide for this important function, the triangular shaped septal gingivae are firmly attached to the teeth nearer to the incisal edges on the proximal sides than on the labial and lingual in order to provide sloping floors for the sulci and to prevent the entrance of food into the inter-dental spaces. This triangular form of the septal gingivae is so essential that every possible provision is made to protect and retain it. The close fitting contact surfaces of the teeth protect these tissues from any harmful pressure of food. There is no provision made for the tissues to withstand the forced entrance of coarse, stiff nylon bristles into these spaces. The need for this exact form of the tissues does not lessen with age.

It is necessary to clean the portions of the teeth that can be seen, but it is much more important to clean portions that are out of sight, such as the contact surfaces. There is no type of brush and no method of using it that can clean these surfaces when the teeth are in a normal position; they must be cleaned some other way. Wide, flat dental floss, when properly controlled, can thoroughly and safely clean these proximal surfaces. The daily care of badly denuded cementum on the proximal sides of the teeth is a much more difficult problem, but very wide, firm dental tape is helpful in most cases. A thin, flat rubber point, with the right degree of firmness, would be helpful in extreme cases. Wood points are too harsh on the tissues.

It is rational to state that no type of toothbrush should be used for cleaning the teeth until the investing tissues are healed and ready to endure it, but there are three things that make any brush too harsh and irritating even for well-healed tissues. The first and most harmful of these, is the use of coarse, stiff bristles that the manufacturer can so easily obtain. The second one is grouping the bristles into large tufts and trimming them into different shapes and lengths. Then, as might be expected, trimming the bristles too short increases their rigidity.

A good brush, that can be used safely and efficiently in all locations in the mouth, should have small tufts of very fine natural bristles that are placed close together in four parallel rows that are trimmed to a proper even length. The flexibility of the bristles permits them to bend and fit all widths and forms of teeth and to enter the interdental spaces as far as the septal gingivae will permit and tolerate. The fourth row of bristles gives the brush a better width for cleaning the lingual side of the anterior teeth.

When this safe type of brush is used, the main strokes of the bristles should be guided to go in the same general direction that the food does during mastication. They should pass upward over the upper teeth and gingival tissues and downward over the lower. That permits the bristles to pass gently through the gingival sulci to thoroughly clean them. When the bristles of the brush travel in the opposite direction, the free margins of the gingivae have the tendency to carry the needed friction past the very portions of the teeth that are most urgently in need of it. Since the gingival tissues are end organs, this physiologic method of using the brush is very beneficial, for it presses out the stagnant blood and allows fresh arterial blood to enter. It gives just the right amount of pressure to stimulate a healthy circulation and to strengthen and toughen the gingivae, but all stimulants become irritants when they pass a certain stage of severity.

Concluding Statement

The outstanding observation to be made from the opinions expressed is that the cleansing technic for the patient to use must be hand tailored to fit his requirements. These individual requirements vary with the mouth size, regularity and inclination of the teeth, amount of recession, especially interdental recession, the tendency for soft deposits and calculus to form, presence or absence of acute ulcerative gingivitis, manual dexterity, age, and desire to coöperate on the part of the patient, and others. A simple child's brushing method will seem to keep the teeth clean and perio-

dontal tissues healthy throughout the lifetime in one patient while in another much time consuming and carefully directed effort will not give so satisfactory a result. Problems increase as the gingiva recedes below the amelo-cemental junction and especially when deep interdental losses lead to large holes between the teeth.

The beautifully healthy gingival and periodontal tissues to be seen in patients of many periodontists is evidently the result of painstaking study of all these requirements and of time spent with the patient to teach the many details of technic which may be necessary. The instruments used to attain these results may vary with the individual requirements but also with the habit experience of the dentist doing the teaching. One dentist has learned to use a small hard bristle brush effectively without damage while another has been trained in the use of a wider softer bristle brush. A thoroughly controlled experimental study might show that one or the other instrument is more economical of time or less dangerous if used improperly, or more easy to keep sanitary, but it now appears that the matter is not as important as many of us had thought. The essential is cleanliness without injury. The method in any given mouth and patient may require several instruments and much detailed instruction in their use. But just as in the case of root surface curettage several kinds of instruments may be used effectively by different operators, so the cleansing instruments used by the patient may vary depending on the ideas of the dentist doing the teaching. A method which keeps the tooth surfaces clean and the gingiva and periodontal tissues in health without appreciable toothbrush trauma recession or abrasion in long periods of years and which does not require an inordinate amount of time or effort on the part of the patient or excessive wear and tear on instruments is good. Such results are being accomplished by more than one type of instrument and method of application. But all methods require study to understand and meet individual requirements and much time to teach the patient the necessary details of care and to establish good habits which will be followed throughout the lifetime.

—Harold J. Leonard

Attention! Members of the A. A. P.

The Committee for the Annual Seminar of the Academy wish to announce the next postgraduate course to be given under the auspices of the Kellogg Foundation at the University of Michigan, Ann Arbor, on May 2, 3, and 4, 1949.

A tentative program has been arranged and consists of the following lecturers:

Dr. John Sheldon, Univ. of Michigan
"Relationship of Allergy to Periodontal Disease."
Dr. Arthur Curtis, Univ. of Michigan
"Oral Manifestations of Dermatology."
Dr. Jerome Conn, Univ. of Michigan
"Diabetes and Endocrine Disturbances."
Dr. C. V. Weller
"Physiological Changes of Aging."
Dr. Joseph P. Weinmann, University of Illinois
"Histopathology of Periodontal Disease."
and
"Concepts of Inflammation."
Dr. Maury Massler, University of Illinois
"Differential Diagnosis."
and
"Bullous Lesions of the Mouth."

This program along with directed round-table discussions and movies will be of considerable value to the periodontist in everyday practice. Only a limited number can be accommodated because of the housing problem at the Michigan Union and you are therefore urged to write Dr. Paul Jeserich at the Kellogg Institute, University of Michigan Dental School, Ann Arbor, Michigan, for an application as soon as possible.

—Harold G. Ray, *Chairman*

Report of Past Presidents' Special Committee

To the Editor of the Journal of Periodontology:

At the breakfast of the Past Presidents of the American Academy of Periodontology, it has been the custom for several years to contribute to a Fund from which awards have been made from time to time to someone who, in the preceding year, had made a notable contribution to dentistry.

At the recent meeting in Chicago, it was the unanimous vote of those present, that the award this year be given to Dr. William J. Gies for his monumental work as editor of the Horace Wells Memorial Volume about to come off the press.

This has been a labor of love on the part of Dr. Gies as has been the innumerable contributions which he has made to the advancement of dentistry since he became its benefactor in 1909.

For more than two years, he has devoted all his time to bringing together and editing the nation-wide material which goes to making up the Wells Memorial Volume. In no better way can we, as members of the dental profession, show our appreciation of the unselfish service which Dr. Gies has rendered in editing and preparing for publication this Wells Volume, than by purchasing a copy. It is a book which should find its way into the hands of every dentist in this country and be given a place in every public library.

As Chairman of the Committee, it was my pleasant duty to transmit to Dr. Gies, the award made by the Past Presidents of the Academy. For their information and for that of the members of the Academy, I am including the letter of acknowledgement by Dr. Gies in this report.

November 29, 1948 —ARTHUR H. MERRITT

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632 W. 168th Street, New York City 32, November 1, 1948

Dr. Arthur H. Merritt,
Chairman of Committee of Past Presidents,
American Academy of Periodontology.

Dear Dr. Merritt:

I thank you heartily for your letter on October 28 from the Past Presidents of the American Academy of Periodontology, and for their award that came with it.

I am deeply impressed by the kindness and generosity which are manifested by the sentiments in your letter, and also by the stated basis for the award. I am very happy to receive these special evidences of interest in the successful outcome of the prolonged, difficult and patient effort to make the Wells Memorial Volume, by careful endeavor, not only a tribute to a great dentist, but also an outstanding credit to the dental profession.

Please state to the Past Presidents of the Academy my very deep appreciation of the high honor their sentiments and award have conferred upon me. Also kindly assure them of my earnest hope that, by good behavior in the future, I may continue to hold the "admiration and esteem" you expressed in your letter.

Yours sincerely,
WILLIAM J. GIES

THE AMERICAN BOARD OF PERIODONTOLOGY

The American Board of Periodontology will hold its examination next summer and fall for certification of dentists expert in the practice of periodontia. The written examination will be held in various cities on July 30th and the oral examination on October 11th in San Francisco or nearby city. Application and case report blanks should be obtained at once by those who desire to take the 1949 examinations. The completed application blank with application fee and photograph should be sent in early this winter and the completed case reports before June 15th. Correspondence should be addressed to Harold J. Leonard, D.D.S., Secy.-Treas., 745 Fifth Avenue, New York 22, N. Y.

Editorials

Dentistry's Good Fortune

AT A Testimonial Dinner in July, 1937 in Atlantic City, many dentists paid honor to a man who has done more to gain respect for dentistry from the other sciences than anyone outside of the profession—Dr. William J. Gies. This was an important occasion with many speakers recounting the various contributions that had been made by Dr. Gies for the advancement of dentistry. Those present were impressed by the modest and clever way in which the guest of honor turned the tribute back to dentistry, by stating that it was the worthy spirit of dentistry that had prompted this appreciation of his personal efforts in its behalf.

Throughout the time that has intervened since that notable event, this friend of dentistry has continued his efforts for its benefit. In recent years, Dr. Gies has applied himself unremittingly to the task of compiling all worthwhile information on Horace Wells and his contribution to mankind. This has required continuous day and night effort for several years which is now culminated in the Wells Memorial Volume of 450 pages. Those who are familiar with the accuracy in details of any work completed by Dr. Gies, can fully appreciate how valuable to dental history this new book will be. The collecting and sifting of a great amount of data and extensive correspondence required infinite patience and laborious effort.

The spirit of dentistry is again challenged. Will its response measure up to the claim? All those who have faith in it believe that it will. Its appreciation will be proved in a very concrete fashion such as a Wells Memorial Volume in every public library put there by dentists, and others ordered for personal use. It is a privilege to be able to express in such a special way one's appreciation of Wells' great benefaction and also for Dr. Gies' accurate record which is an expression of the latter's faith in dentistry. (*See* p. 42.)

—G.R.S.

Financing Dental Journals

THERE are three principal ways of financing dental journals, by: (1) income from advertisements (2) adequate subscription price (3) the owner's ability and willingness to pay the expense. Lately, the increased cost of printing has made it difficult, and in some instances financially impossible, for a dental society to publish its journal without an unusually large circulation.

The Journal of Periodontology has been financed by its owner, the American Academy of Periodontology, and by its members since 1930. It is now in its 20th Volume. Although the number of subscribers is small its breadth of circulation is large, since it has subscriptions in many foreign countries.

However, the time has come when it is essential to increase the circulation of this Journal, not alone to assure its continuation, but to spread the science of periodontology more effectually.

The study of periodontology is not just a matter of mastering a single technic in the treatment of periodontal disease. It should include many related sciences. A familiarity with its development from the earliest times is desirable. This Journal aims to keep before the reader the earliest contributions to the development of periodontia as we now know it. It carries articles of research in periodontology and in related sciences, reports of research in nutrition and dental health, also articles and discussions on the practice of periodontia. It is conferring a favor on a confrere to tell him about this Journal.

The President of the Academy is asking each member to get one new subscriber for 1949. To make this as convenient as possible, a subscription envelope is included in each journal sent to members in January. It will help the Secretary if checks are made out to the "Journal of Periodontology."

—G.R.S.

Home Care of the Mouth

AT AN annual meeting of the American Academy of Periodontology, in 1920, one essayist (Dr. Justin D. Towner) recommended using the term, "mouth brush" instead of "toothbrush". The idea is thought-provoking and might be the solution for differences of opinion on details of home care technics.

In the current discussion in the "In Our Opinion" department of this Journal progress is noted, in that the subject of Home Care is being considered in relation to basic principles, rather than particulars. The services of the periodontist is more dependent on continued and effectual home care of the mouth than those of other dentists. For this reason, periodontists have given the subject serious attention. It is possible that also on this account exaggerated consideration has been given to details, glorifying their importance. For the purpose of discussion, it is wiser to keep to major issues if one expects to reach an agreement, or at least an adjustment of opinions.

The January, 1949 issue of the J.A.D.A. contains a Questionnaire Survey on Dental Health Habits—5,480 families were contacted, 80 per cent returning the questionnaires. The survey is especially interesting in conjunction with this Journal's running discussion on Home Technics for the Care of the Teeth.

—G.R.S.

Notice Concerning Horace Wells' Memorial Volume

The Horace Wells Memorial Volume—450 pages—will be ready for sale and distribution soon.

Title—"Horace Wells, dentist; father of surgical anesthesia"

The Editor, Dr. William J. Gies, M.S., Ph.D., Sc.D., L.L.D., F.A.C.D., of Columbia University.

The Volume presents the "Proceedings of centenary commemorations of Wells' discovery in 1844 and lists of Wells Memorabilia, including bibliographies, memorials and testimonials".

The Introduction is the outcome of a thorough study, by the editor, of the records of the "anesthesia controversy". It shows—by elimination of irrelevant and misleading personal and emotional details that have beclouded the "anesthesia story"—a true and unbiased picture of the great discovery.

Successive sections contain complete and detailed accounts—all from local authoritative sources—of the 500 Wells Centenary commemorations in England, France, fourteen nations in Central and South America, and in the states in the United States; and, for this country, present not only records of the celebrations under the auspices of state and district societies and various local organizations but also of the commemorations in the U. S. Army commands, U. S. Navy districts, and U. S. Public Health stations.

This Volume, as a centenary commemoration, preserves and clarifies the abiding truth in the history of the origination and evolution of general surgical anesthesia. In honoring "Horace Wells, dentist and father of it all", the book portrays the basic realities of this great tradition, which is an inspiration for all dentists and a source of profound thankfulness for all mankind.

Our slogan—A Wells Volume In Every Library.

Why not order a Volume for your home town library now?

The book will be sold at cost, and the estimated price will be between $4.00 and $5.00.

DR. EUGENE M. CLIFFORD,
Chairman, Horace Wells Centenary Committee,
57 Pratt Street,
Hartford, Connecticut.

Dr. Clifford:

Please enter my order for ..... copies of the Horace Wells Memorial Volume.

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THE AMERICAN BOARD OF ORAL PATHOLOGY

As its Sponsoring Agency the American Academy of Oral Pathology announces the formation of the American Board of Oral Pathology. This board is incorporated in the District of Columbia and has been organized in accordance with the policies established by the Council on Dental Education of the American Dental Association. The members of the board are:

LESTER CAHN, *New York City* ..... President
HAMILTON B. G. ROBINSON, *Columbus, Ohio* ..... Vice-President
LT. COL. JOSEPH L. BERNIER, *Washington, D. C.* ..... Secretary-Treasurer

The Board of Directors consist of the following, in addition to those presently serving as members of the board:

KURT H. THOMA, *Boston, Massachusetts*
PAUL E. BOYLE, *Philadelphia, Pennsylvania*
DONALD A. KERR, *Ann Arbor, Michigan*
HENRY M. GOLDMAN, *Boston, Massachusetts*

Detailed information as to eligibility for certification and character of the examination for such certification may be obtained by writing the Secretary—

LT. COLONEL JOSEPH L. BERNIER, D.C.
Army Institute of Pathology
Washington 25, D. C.

The board will conduct its first examination in the fall of 1949.

Postgraduate Study Courses

The following courses in Periodontology are scheduled in the postgraduate division at Tufts College Dental School. Periodontal courses are given under the direction of Dr. Irving Glickman.

A. Sixteen weeks, full time, from January 31, to May 28, 1949.
B. One day per week for twelve weeks, each Monday from January 17 to April 11, 1949.
C. Five days, May 16 to May 21, 1949. This course is devoted to the Kirkland gingivectomy technique and is given by Dr. Olin Kirkland.
D. Five days, June 13 to June 17, 1949.

All courses listed above are participating courses. One half of the time is devoted to patient management and treatment. Information concerning other postgraduate activities may be obtained by writing to the Director of Graduate and Postgraduate Studies, Tufts College Dental School, 416 Huntington Avenue, Boston, Massachusetts.

Two courses in Periodontology are scheduled to commence at the University of Toronto January 5 and May 3, 1949. Included in the topics to be reviewed will be a general consideration of the nature of periodontal disease, examination and case management, pocket therapy, oxygen therapy, metabolic influences on the periodontium, significance of occlusal equilibration, gingival recession, pocket surgery, application of principles of periodontal practice to operative and prosthetic procedure. The courses will be under the general direction of Drs. H. K. Box, and C. H. M. Williams.

Those interested are asked to inquire immediately respecting enrollment in these classes as there are only a few vacancies remaining. Please address inquiries to: The Dean, Faculty of Dentistry, University of Toronto, 230 College Street, Toronto 2B, Canada.

MID-ATLANTIC STATES SEMINAR IN ORAL MEDICINE

May 22-27, 1949

Offered by the Postgraduate Division, University of Pennsylvania, School of Dentistry, Skytop Lodge in the Pocono Mountains, Pennsylvania

Regional seminars in Dental and Oral Medicine have been held in the west and middle west for many years. They have not only stimulated the practitioner's interest in this increasingly important phase of dental practice but they have served to bring together clinicians and investigators of many branches of the health service professions related to dentistry. This opportunity for

a free interchange of ideas and questions among the dental practitioners, clinicians, and investigators has resulted in a broadened concept of the health service opportunities and responsibilities of the dentist and it has also suggested fertile fields for future research and investigation.

The general theme of this Mid-Atlantic States Seminar in Oral Medicine is the clinical application of the biologic and medical sciences.

Lecturers: J. L. T. Appleton, B.S., D.D.S., Sc.D.; Oscar V. Batson, M.A., M.D.; Herman Becks, M.D., D.D.S.; Leon H. Collins, Jr., A.B., M.D.; Julius H. Comroe, Jr., A.B., M.D.; Thomas J. Hill, D.D.S.; Samuel Charles Miller, D.D.S.; Harold F. Robertson, Jr., M.D.; Kurt H. Thoma, D.M.D.,

Write for information to Dr. L. W. Burket, Director, Postgraduate Division, School of Dentistry, University of Pennsylvania, 4001 Spruce Street, Philadelphia 4, Pennsylvania. Registration fee includes lodging and meals.

The American Academy of Oral Pathology will hold its annual meeting at the Stevens Hotel, Chicago, Illinois on February 6, in Private Dining Room 2. Discussions of various phases of Oral Pathology will be presented by members and guests. A Banquet will be served on Sunday evening, February 6, in the same room. All interested dentists, physicians and pathologists are invited to participate in the meeting.

HAMILTON B. G. ROBINSON, *Editor*
American Academy of Oral Pathology

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